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Original article

The effect of open kinetic chain knee extensor resistance training at different training loads on anterior knee laxity in the uninjured

Massimo G. Barcellona [a], Matthew C. Morrissey [b, *]

[a] Academic Department of Physiotherapy, Faculty of Life Sciences & Medicine, King's College London, UK
[b] Faculty of Health Sciences, University of Ljubljana, Slovenia

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ABSTRACT

Background: The commonly used open kinetic chain knee extensor (OKCKE) exercise loads the sagittal restraints to knee anterior tibial translation.
Objective: To investigate the effect of different loads of OKCKE resistance training on anterior knee laxity (AKL) in the uninjured knee.
Study design: non-clinical trial.
Methods: Randomization into one of three supervised training groups occurred with training 3 times per week for 12 weeks. Subjects in the LOW and HIGH groups performed OKCKE resistance training at loads of 2 sets of 20 repetition maximum (RM) and 20 sets of 2RM, respectively. Subjects in the isokinetic training group (ISOK) performed isokinetic OKCKE resistance training using 2 sets of 20 maximal efforts. AKL was measured using the KT2000 arthrometer with concurrent measurement of lateral hamstrings muscle activity at baseline, 6 weeks and 12 weeks.
Results: Twenty six subjects participated (LOW n = 9, HIGH n = 10, ISOK n = 7). The main finding from this study is that a 12-week OKCKE resistance training programme at loads of 20 sets of 2RM, leads to an increase in manual maximal AKL.
Conclusions: OKCKE resistance training at high loads (20 sets of 2RM) increases AKL while low load OKCKE resistance training (2 sets of 20RM) and isokinetic OKCKE resistance training at 2 sets of 20RM does not.

1. Introduction

The anterior cruciate ligament (ACL) is the primary restraint to anterior translation of the tibia on the femur (Markolf et al., 1976; Shoemaker and Markolf, 1985), providing greater than 80% of the passive restraint to this movement (Butler et al., 1980). Injury of this important ligament leads to loss of function (Noyes et al., 1991) and to functionally disabling secondary complications including meniscal damage (Tayton et al., 2009), articular cartilage defects (Hanypsiak et al., 2008) and the early onset of knee joint osteoarthritis (Daniel et al., 1994; Lohmander et al., 2004; Pinczewski et al., 2007; Oiestad et al., 2009; Keays et al., 2010). Given that ACL injury (ACLI) has been shown to lead to knee joint osteoarthritis regardless of conservative or surgical management (Lohmander et al., 2004; Meuffels et al., 2009; Myklebust et al., 2003; von Porat et al., 2004) it has been proposed that the best way to manage this injury is to prevent it (Bahr, 2009). Recent studies have reported that specific exercise interventions can reduce the risk of ACLI (Mandelbaum et al., 2005; Gilchrist et al., 2008; Pasanen et al., 2008). These studies focused on the neuromuscular components of the training programmes.

There are no studies evaluating the effect of specific exercise interventions for modifying knee joint laxity in the uninjured despite this variable being a significant risk factor for ACLI (Uhorchak et al., 2003; Myer et al., 2008; Vauhnik et al., 2008). The principal reason for this is that many researchers appear to suggest that knee laxity is a non-modifiable risk factor for ACLI (Alentorn-Geli et al., 2009). Despite this view there is evidence in the literature to suggest that individuals performing certain types of sports or activities may have reduced knee joint laxity when compared to controls or individuals partaking in different sports or activities (Kettunen et al., 1997; Rosene and Fogarty, 1999; Ng and Maitland, 2001; Ergun et al., 2004). Among the possible reasons for this finding is the possibility that loading of the passive restraints to

* Corresponding author.
E-mail address: morrissey.matt@ymail.com (M.C. Morrissey).

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sagittal translation of the tibia at the knee, as a result of certain activities or sports will lead to a reduction in knee joint laxity over time. This suggestion is based on the scientific literature regarding the response of soft tissues to loading (Woo et al., 1987, 2006) and on in vitro data showing that the uninjured ACL will respond to mechanical loading via an increase in stiffness and strength (Cabaud et al., 1980) and an increase in collagen expression (Hsieh et al., 2000, 2002; Kim et al., 2002; Tetsunaga et al., 2009); as will the other soft tissues at the knee (Kjaer, 2004).

Given that OKCKE exercise has been shown to mechanically load the ACL, and given that the magnitude of ACL loading with this exercise is dependent on the load applied to the tibia (Beynnon et al., 1992, 1995, 1997), this exercise provides a way to impart different loads on the ACL. In the ACL-injured knee, two studies support OKCKE training's ability to decrease AKL (Morrissey et al., 2009; Barcellona et al., 2014a). In the first of these, a negative correlation was found between the load used in OKCKE and AKL increases in the ACL-injured and ACL-reconstructed knee (Morrissey et al., 2009). In the more recent study, AKL decreased after ACLI in low load (2 sets of 20 repetition maximum) but not high load (20 sets of 2 repetition maximum) OKCKE training (Barcellona et al., 2014a). The apparent discrepancy in the findings of these two studies (Morrissey et al., 2009; Barcellona et al., 2014a) may be due to there being a negative correlation between OKCKE resistance training and AKL change up to a certain point, and that additional loads may then lead to tissue lengthening; implying a non-linear bell-shaped relationship. It is, therefore, important to study the effect of OKCKE exercise across the load spectrum (Barcellona et al., 2014a).

The main aim of this study was to assess the effect of different loads and types (isotonic and isokinetic) of OKCKE resistance training exercise on AKL change in the uninjured knee. The main hypothesis tested in this study was that the use of higher loads of OKCKE muscle resistance training would lead to a greater reduction of AKL in the uninjured knee when compared to lower loads of this exercise.

2. Methods

This study was a prospective, single-blind randomised controlled trial with repeat measures at baseline and after 6 weeks and 12 weeks of training (Fig. 1). Ethical approval for the study was granted by the King's College London Research Ethics Committee (CREC/06/07-138). A convenience sample of subjects was recruited from the Guy's Campus of King's College London and the surrounding area via emails and posters distributed locally. Prior to the commencement of baseline testing subjects were given time to read the study information sheet. They were asked if they had any questions about their participation, and following a verbal explanation of the study procedures, they read and signed a consent form. Subjects then completed a general questionnaire consisting of questions regarding personal details, history of relevant injury to either lower limb, current level of sports participation and leg dominance. Current level of sporting activity was also assessed by asking subjects to rate themselves on the Tegner Activity Scale (Tegner and Lysholm, 1985).

Subjects were deemed suitable for inclusion in the study if they: i) were aged 18–60 years and ii) did not have a greater than 3 mm side-to-side difference in AKL. Subjects were excluded from participating if they: i) had neurological, systemic rheumatological or muscular diseases and ii) had pathology or traumatic injury to either lower limb within the previous year.

Anthropometric measurements including body mass, height, knee range of motion and girth were recorded – from the right and then the left sides, where applicable – with standard equipment and with the subject lying supine.

AKL was assessed by one examiner (MB) – blinded to group allocation – with the KT2000 arthrometer (Medmetric Corp., San Diego, USA). The procedures are described in Barcellona et al., (Barcellona et al., 2014b) and included surface electromyography (EMG) measurement of the lateral hamstrings during AKL testing; recorded using the Delsys Bagnoli-4 System with DE-2.1 Single Differential Electrodes (Delsys Inc., Boston, MA). Three maximal voluntary isometric contractions (MVIC) of the knee flexors were performed, prior to laxity testing, against the resistance applied by the principal examiner in a seated position on the edge of a plinth with the hip and knee at 90°. Verbal instructions were standardised.

A 133 N and a manual maximal anterior knee laxity test was conducted for each leg; consisting of three anterior and posterior repetitions at the 88 N force (applied via the handle of the KT2000 arthrometer and denoted by an audible beep) followed by 5 repetitions of the particular test.

Laxity and EMG data signals were sampled at a frequency of 2000 Hz using a National Instruments USB-6210 portable analogue to digital converter (National Instruments Corporation, Texas, USA) and Labview SignalExpress 2.5.0 software (National Instruments Corporation, Texas, USA).

2.1. Training protocol

Following baseline testing subjects were block randomised (blocks of 6 assignments) to one of three OKCKE training groups. The leg to be trained was chosen at random and the other leg acted as a control. Each subject was invited to attend for training on a three session per week basis for 12 weeks.

For all three OKCKE groups training was carried out between 90° and 0° of knee flexion with a 20 s rest interval between sets. Group 1 (LOW) performed 2 sets of 20 repetition maximum (RM) OKCKE exercise using a standard knee extension device (Health and Leisure, Walthamstow, U.K.). Group 2 (HIGH) performed 20 sets of 2RM with the same device as Group 1. Group 3 (ISOK) performed isokinetic concentric and eccentric OKCKE exercise at 60°/s on the Kin Com Dynamometer (Chattecx, Chattanooga, Tennessee, USA) for 20 sets of 2RM. Subjects in the LOW and the HIGH groups were encouraged to perform each exercise repetition at an average speed of 60°/s, and this was achieved by the use of a metronome. The high dosage performed by the HIGH load group – reflected in the number of sets – was chosen to ensure that a wide spectrum of training dosage was investigated.

For the LOW and the HIGH groups the number of sessions, sets, repetitions and load (kg) was recorded. If subjects in the LOW or HIGH groups performed repetitions above or below the prescribed repetition maximum the weight was adjusted in order to bring the number of repetitions to the desired level.

2.2. Data reduction & analysis

Knee laxity and surface EMG data were analysed as described in Barcellona et al. (Barcellona et al., 2014a) In summary, laxity and EMG data processing was performed using a specifically designed program in Matlab (The Mathworks Inc., Natick, MA, USA) in order to: i) determine a zero displacement point for each of the 5 trials, ii) find the peak displacement for each trial, and iii) compute displacement for each trial to the nearest 0.01 mm. The EMG data was 50 Hz notch and Butterworth band pass filtered (15 Hz–500 Hz). At the 5 points of peak laxity the mean of the root mean square (RMS) of the EMG signal was calculated using five