Altered knee joint neuromuscular control during landing from a jump in 10–15 year old children with Generalised Joint Hypermobility. A substudy of the CHAMPS-study Denmark

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Abstract
Generalised Joint Hypermobility (GJH) is considered an intrinsic risk factor for knee injuries. Knee neuromuscular control during landing may be altered in GJH due to reduced passive stability. The aim was to identify differences in knee neuromuscular control during landing of the Single-Leg-Hop-for-Distance test (SLHD) in 25 children with GJH compared to 29 children without GJH (controls), all 10–15 years. Inclusion criteria for GJH: Beighton score \( \geq 5/9 \) and minimum one hypermobile knee. EMG was recorded from the quadriceps, the hamstring and the calf muscles, presented relative to Maximum Voluntary Electrical activity (MVE).

There was no difference in jump length between groups. Before landing, GJH had 33% lower Semitendinosus, but 32% higher Gastrocnemius Medialis activity and 39% higher co-contraction of the lateral knee muscles, than controls. After landing, GJH had 36% lower Semitendinosus activity than controls, all significant findings.

Although the groups performed equally in SLHD, GJH had a Gastrocnemius Medialis dominated neuromuscular strategy before landing, plausibly caused by reduced Semitendinosus activity. Reduced Semitendinosus activity was seen in GJH after landing, but with no compensatory Gastrocnemius Medialis activity. Reduced pre and post-activation of the Semitendinosus may present a risk factor for traumatic knee injuries as ACL ruptures in GJH with knee hypermobility.

1. Introduction

Biomechanical factors such as knee joint laxity is considered an intrinsic risk factor for traumatic knee joint injuries, e.g. Anterior Cruciate Ligament (ACL) ruptures (Uhorchak et al., 2003; Myer et al., 2008). Individuals with Generalised Joint Hypermobility (GJH) frequently experience knee joint hypermobility or laxity, which is presumed to be an intrinsic risk factor for ACL and other knee injuries in both adults and adolescents (Ostenberg and Roos, 2000; Uhorchak et al., 2003; Ramesh et al., 2005; Myer et al., 2008; Pacey et al., 2010). However, the contribution of GJH and specifically knee joint hypermobility, to the mechanisms behind knee joint injuries is unknown, partly due to a lack of mechanistic studies and inconsistencies in diagnostic tests and criteria for GJH.

GJH is a hereditary condition, which is characterised by increased range of motion due to increased laxity of the connective tissues compared with the normal population. This results in decreased stiffness and stability of the passive structures like joint capsules and ligaments (Grahame, 1999). From a functional perspective, one possible compensation strategy for reduced passive knee joint stability may be increased muscle activation to increase the active stability of the knee joint (Shultz et al., 2004; Hewett et al., 2005). However, it is currently unknown whether a compensation strategy is present during challenging tasks demanding dynamic knee stability, like landing from a jump.

Pre-activation of the stabilizing muscles may increase the dynamic stability of the lower extremity during impact in
challenging tasks (Rozzi et al., 1999; Hewett et al., 2005). Thus, adequate knee joint neuromuscular coordination and control become crucial, since ineffective muscle recruitment may result in knee positioning with increased ACL strain, thereby increasing the risk of injury (Rozzi et al., 1999; Hewett et al., 2005).

Only few studies of muscle activity in individuals with GJH have been performed (Greenwood et al., 2011a,b; Jensen et al., 2013; Smith et al., 2013). One study of children (10-year old) with GJH and at least one hypermobile knee found neuromuscular coordination and control strategies to differ from controls. This was seen during a submaximal isometric knee flexion task, where hamstring muscle activity was reduced. During the same task, knee muscle co-activation ratio was increased, which was suggested to be a compensatory strategy for the lower hamstrings activity (Jensen et al., 2013). Furthermore, decreased maximum isokinetic knee extension and flexion strength were seen in 10-year old girls and women with GJH and also, decreased knee strength balance (Hamstring/Quadriceps ratio) was seen in adults with GJH (Juul-Kristensen et al., 2012). However, these results were all obtained from the entire cohort along with other tests one month prior to the current study. In total, 56 children were recruited, 26 with GJH and 30 controls, matched by age and sex at a group level.

Inclusion criteria for children with GJH were a BT score (Junge et al., 2013) of >5/9, at least one hypermobile knee, and positive standing knee hyperextension confirmed by using a goniometer during supine lying (Ramesh et al., 2005; Myer et al., 2008). Inclusion criteria for controls were a BT score of no higher than 1/9 and no hypermobile knee.

Exclusion criteria for both groups were current pain in the back or lower extremities affecting the ability to jump, previous or current knee trauma, hereditary diseases like Ehlers-Danlos Syndrome, Marfan Syndrome, Osteogenesis Imperfecta and body mass index > 25. Information about previous injuries was obtained from the CHAMPS-study (Wedderkopp et al., 2012).

The Regional Scientific Ethics Committee for Southern Denmark approved the experimental protocol (jnr. S-20080047 HJD/csf) and the study was reported to the Danish Data Protection Agency. Written and oral information about participation in the study was provided to the parents or guardians of the participating child according to the Declaration of Helsinki. Written informed consent for participation was received, and on the day of testing, each child verbally confirmed participation.

2.3. Outcome measures

A standardised test protocol was strictly followed for each child. All testers, except the lead tester, were unaware of each child’s status of GJH or control during the study. The lead tester decided the test leg of the child, defined as the leg with the most hypermobile knee joint for the children with GJH, while the test leg for the controls was selected at random. A 10-min standardised warm-up was completed for each child before the SLHD test.

2.3.1. SLHD test

The SLHD test was modified slightly from the original version describing the arms to be held behind the back (Tegner et al., 1986). The child was asked to jump on the test leg as far as possible allowing arm swing assistance and to land standing steadily on the test leg for at least 2–3 s. The child had one practice trial and then three SLHD tests and additional jumps, until no further progress in jump length was observed. Between each test the child had a 30 s rest. The longest jump, measured in cm from the toe in the starting position to the backside of the heel in the landing position, was used for analysis. In a pilot study, reproducibility for SLHD for children aged 11 and 14 years had ICC values for inter-session of 0.93 respectively 0.84 (unpublished data).

2.3.2. Sport participation

The weekly amount of organised sports activity was registered by SMS surveys every week. The SMS question to the parents of the single child was: “How many times did your child participate in organised sports within the last week?” with the possibility to type the relative number between 0 and 8, with 8 meaning more than 7 times. The individual child’s mean amount of organised sports activity during two months was used for analysis.

2.3.3. Maximum voluntary contraction

Maximum voluntary contraction (MVC) for knee flexion and extension was measured during sitting with a straight back without support, the hips at 90° flexion, the knees at 60° flexion and both arms placed across the chest (Thorborg et al., 2013). The moment arm for knee flexion and extension was measured as the distance between the centre of rotation of the knee joint and a line projected perpendicular to the direction of force applied just proximal to the lateral malleolus.
For measuring ankle plantar flexion MVC, the children were supine on an examination bench with an extended knee and the ankle placed in 10° dorsal flexion, free of the bench. To minimise trunk and hip movement during the test, a tester stabilized the hip. A strap connected to the force transducer was positioned around the foot (Fig. 1). The moment arm for ankle plantar flexion was measured as the distance from the medial malleolus (taken as the indication of the centre of ankle joint rotation) perpendicular to the line of force application at the middle of the foot strap. Three maximum attempts were performed for measuring MVC with 1 min rest in between. The testers verbally encouraged the child during each MVC.

MVC was measured with a strain-gauge force transducer (Nobel Load Cells KJ2-2 2 kN, England) expressed in Nm and normalised to body mass (Nm/kg) for knee extension, knee flexion and ankle plantar flexion. Further, isometric strength ratios of maximum knee flexion and knee extension (KF/KE) as well as the ratio of maximum plantar flexion and knee extension (PF/KE) were calculated, since the PF further have knee flexor function similar to the KF.

2.3.4. Electromyography

Bipolar surface electromyography (EMG) signals from the knee flexor and knee extensor muscles (Gastrocnemius Medialis: GM; Gastrocnemius lateralis: GL; Semitendinosus: ST; Biceps femoris: BF; Quadriceps–vastus medialis: VM; Quadriceps–vastus lateralis: VL) were measured during MVC and during the SLHD test.

Prior to testing, skin preparation procedures included hair removal, light abrasion and disinfection. Electrode placement and orientation was positioned according to SENIAM recommendations (SENIAM). The Ag/AgCl EMG electrode (Blue sensor N, Ambu Denmark) had a pre-gelled diameter of 10 mm and an inter-electrode distance of 2 cm. An accelerometer was positioned over the trochanter major for definition of the landing. All electrodes and cables were subsequently attached to the leg with elastic bands to keep them properly fixed to the skin. To control for cross talk between the ST and the BF muscles, we used the recommended electrode position from SENIAM and carefully chose a short inter-electrode distance to be as selective as possible on each muscle. Furthermore, we performed a visual inspection of the simultaneous signal from the two muscles to make sure that the two adjacent muscles showed distinct activation in different standard tasks indicating a minimum of cross talk.

The EMG signal and acceleration were sampled via a telemetry EMG system (Telemyo DTS and Telemyo mini receiver, Noraxon U.S.A. Inc.) through integration with a computer equipped with data collection software (MyoResearch xp master, Noraxon U.S.A. Inc.). The EMG signal was A/D converted and sampled at 1500 Hz using the Telemyo DTS system with low-pass cut-off filtering at 500 Hz and a 1st order high-pass at 10 Hz. EMG signals were amplified with a total gain of 500 Hz. Accelerometer data were sampled at 1500 Hz with a low-pass cut off at 50 Hz. The sensitivity was ±0.67 V/g.

Landing during SLHD was defined as the time point where the acceleration exceeded 5g. Maximal Voluntary Electrical activity (MVE) for each muscle was defined as the highest EMG activity measured as the root mean square (RMS) amplitude in a 100 ms moving window across the whole MVC. EMG activity was calculated 100 ms before and 50 ms after landing of SLHD as RMS amplitude, and for each muscle normalised to MVE of the relevant test and presented as %MVE (Fig. 2).

Time of onset for muscle activity in ms was defined, for each muscle, relative to landing time, determined by the accelerometer. An increase in muscle activity was defined as the signal exceeding a set trigger level of 2.5% of maximum EMG during each jump in a 20 ms window, identified by an algorithm. Afterwards, this time of onset was evaluated by visual inspection, previously considered a valid evaluation of EMG activation characteristics (Hodges and Bui, 1996).

A muscle co-contraction index (CCI), defined as the simultaneous activation of two muscles (Rudolph et al., 2001), was calculated cumulative for the Quadriceps (Q) and Hamstring (H) groups (H/Q). Also, we divided the single medial or lateral knee muscles of the latter muscle groups as well as the Gastrocnemius muscle group combined in indices of two: Quadriceps and Hamstring, Q–H (VL–BF and VM–ST), Quadriceps and Gastrocnemius, Q–G (VL–GL and VM–GM), and the Hamstring and Gastrocnemius muscles, H–G (ST–GM and BF–GL). Co-contraction was determined using the following

Fig. 1. Testing set-up for Maximal isometric Voluntary Contraction (MVC) for ankle plantar flexion.

Fig. 2. Example of EMG recordings in mV of the six investigated muscles: Quadriceps vastus lateralis: VLO; Quadriceps – vastus medialis: VMO; Semitendinosus: SEM; Biceps femoris: BIC; Gastrocnemius Medialis: MED; Gastrocnemius lateralis: LAT. Top line is the accelerometer data indicating impact of landing with the vertical line. The time frames indicate 100 ms before landing and 50 ms after landing.
equation: & \text{MVEmin} = \text{MVEmax} \times \left( \frac{\text{MVEmin} + \text{MVEmax}}{2} \right) \text{ (Rudolph et al., 2001).} \text{ MVEmin was the level of activity in the less active muscle and MVEmax was the level of activity in the more active muscle before landing. This index was multiplied by the total activity of the two muscles, providing an estimate of the relative simultaneous activation of the suggested agonist and antagonist, as well as the magnitude of the co-contraction (Greenwood et al., 2011a,b).}

2.4. Statistical analysis

The outcome variables were normally distributed. An un-paired t-test was used to compare mean age, height, weight, sports participation and jump length between groups.

Group differences for MVC and MVC ratios, relative EMG activity level, CCI and time of onset were analysed with multi-level linear regression adjusted for sex, grade, height, weight and sports participation. Grade was used as a proxy for age in the analyses.

Due to the exploratory nature of the current study, no adjustments for multiple testing were applied. & 

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2.5. Results

A total of 56 children were recruited for this study, but on the day of testing, two of the children declined to participate. Fifty-four children participated in this study, but as there was no sex difference in any of the results, the data from the boys were kept in the analysis.

The groups were comparable on demographics (age, height, weight, sports participation) (Table 1).

There was no difference between groups in performance of the SLHD test (Table 2).

Furthermore, no between-group differences were seen for isometric muscle strength in ankle plantar flexion, knee extension or knee flexion. Similarly, no group differences were found for isometric strength ratios of KF/KE or PF/KE (Table 2).

Before landing, GJH activated ST significantly less than controls, corresponding to 33% lower activity. GM was activated significantly more before landing for GJH than controls, corresponding to 32% higher activity (Table 3). After landing, GJH activated ST significantly less than controls, corresponding to 36% lower activity (Table 3). No other differences were observed.

For time of onset prior to landing, there was a tendency for GJH to activate ST 31% earlier and a tendency to activate VM 13% later than controls (Table 3). There was no group difference in time of onset after landing (not shown in tables). A significantly higher CCI for GJH than for controls was observed before landing for the lateral knee muscle group of Quadriceps–Gastrocnemius (VL–GL), corresponding to a 39% higher CCI. There was no group difference in any other CCI: cumulative Hamstring/Quadriceps (H/Q), Quadriceps–Hamstring (VL–BF, VM–ST), Quadriceps–Gastrocnemius (VM–GM), Hamstring–Gastrocnemius (ST–GM, BF–GL) (Table 4).

4. Discussion

The main finding of the current study was that although no difference in jump length was found between groups, children with GJH used a different knee neuromuscular strategy than controls before and after landing from the SLHD test. Generally, ST was activated less in children with GJH than in controls, both before and after landing from the SLHD test. At the same time, an increased activation of GM and a larger CCI of the lateral knee muscle group (VL–GL) was seen for the GJH group before landing, while no increased GM activity was seen after landing.

Both groups performed equally in the SLHD test. However, this was apparently achieved by a neuromuscular strategy relying on higher GM activity in the GJH group to compensate for reduced ST muscle activity compared with controls. The findings of the current study are in line with the results from Jensen et al., where children aged 10 years with GJH and at least one hypermobile knee

<table>
<thead>
<tr>
<th>Group</th>
<th>GJH (n = 25)</th>
<th>Controls (n = 29)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brighton score (0–9)</td>
<td>Median 6</td>
<td>Median 0</td>
<td>0.001</td>
</tr>
<tr>
<td>Age (years)</td>
<td>11.5 ± 1.3</td>
<td>11.6 ± 1.1</td>
<td>0.69</td>
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<tr>
<td>Height (cm)</td>
<td>154 ± 10.3</td>
<td>153.8 ± 9.3</td>
<td>0.85</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>43.7 ± 10.2</td>
<td>41.7 ± 6.6</td>
<td>0.39</td>
</tr>
<tr>
<td>Sports participation (h/week)</td>
<td>2.3 ± 1.9</td>
<td>2.6 ± 1.9</td>
<td>0.35</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Group</th>
<th>GJH</th>
<th>Controls</th>
<th>p-value</th>
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</thead>
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<tr>
<td>Performance test</td>
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<tr>
<td>SLHD (cm)</td>
<td>120.8 ± 18.2</td>
<td>118.7 ± 18.2</td>
<td>0.67</td>
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<tr>
<td>Isometric muscle strength</td>
<td></td>
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<tr>
<td>Ankle plantar flexion (Nm/kg)</td>
<td>1.09 (0.8–1.3)</td>
<td>1.27 (1.1–1.4)</td>
<td>0.26</td>
</tr>
<tr>
<td>Knee flexion (Nm/kg)</td>
<td>5.66 (4.7–6.5)</td>
<td>5.41 (4.5–6.2)</td>
<td>0.62</td>
</tr>
<tr>
<td>Knee extension (Nm/kg)</td>
<td>5.11 (4.1–6.1)</td>
<td>4.84 (3.9–5.7)</td>
<td>0.08</td>
</tr>
<tr>
<td>KF/KE ratio</td>
<td>1.22 (1.1–1.4)</td>
<td>1.22 (1.1–1.3)</td>
<td>0.99</td>
</tr>
<tr>
<td>PF/KE ratio</td>
<td>0.22 (0.1–0.2)</td>
<td>0.28 (0.2–0.3)</td>
<td>0.11</td>
</tr>
</tbody>
</table>

<table>
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<tr>
<th>Group</th>
<th>GJH</th>
<th>Controls</th>
<th>p-value</th>
</tr>
</thead>
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<tr>
<td>Pre activation (MVE)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VM</td>
<td>28.6 (22.2–34.9)</td>
<td>26.7 (20.7–32.8)</td>
<td>0.68</td>
</tr>
<tr>
<td>VL</td>
<td>25.83 (19.8–31.7)</td>
<td>28.57 (22.9–34.1)</td>
<td>0.50</td>
</tr>
<tr>
<td>GM</td>
<td>32.52 (26.5–38.5)</td>
<td>22.17 (16.5–27.8)</td>
<td>0.01</td>
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<td>GL</td>
<td>25.56 (19.2–31.8)</td>
<td>28.67 (22.7–34.6)</td>
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<tr>
<td>BF</td>
<td>26.64 (20.1–33.2)</td>
<td>26.55 (20.4–32.6)</td>
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<tr>
<td>ST</td>
<td>21.64 (15.4–27.8)</td>
<td>32.29 (26.3–38.2)</td>
<td>0.01</td>
</tr>
<tr>
<td>Post activation (MVE)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VM</td>
<td>27.03 (20.7–33.3)</td>
<td>24.04 (18.1–29.9)</td>
<td>0.49</td>
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<tr>
<td>VL</td>
<td>27.69 (21.4–33.9)</td>
<td>23.55 (17.6–29.4)</td>
<td>0.16</td>
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<tr>
<td>GM</td>
<td>26.09 (19.6–32.4)</td>
<td>25.18 (19.1–31.3)</td>
<td>0.84</td>
</tr>
<tr>
<td>GL</td>
<td>27.61 (21.3–33.8)</td>
<td>24.01 (18.1–30.0)</td>
<td>0.41</td>
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<tr>
<td>BF</td>
<td>24.75 (18.5–30.9)</td>
<td>26.34 (20.3–32.3)</td>
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<tr>
<td>ST</td>
<td>18.71 (13.1–24.2)</td>
<td>29.31 (23.8–34.7)</td>
<td>0.01</td>
</tr>
<tr>
<td>Time of onset relative to landing (ms)</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>VM</td>
<td>−13 (−14; −11)</td>
<td>−15 (−16; −13)</td>
<td>0.06</td>
</tr>
<tr>
<td>VL</td>
<td>−11 (−13; −10)</td>
<td>−13 (−14; −11)</td>
<td>0.20</td>
</tr>
<tr>
<td>GM</td>
<td>−12 (−13; −11)</td>
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<td>0.24</td>
</tr>
<tr>
<td>GL</td>
<td>−12 (−13; −11)</td>
<td>−12 (−13; −11)</td>
<td>0.38</td>
</tr>
<tr>
<td>BF</td>
<td>−15 (−16; −15)</td>
<td>−15 (−16; −14)</td>
<td>0.70</td>
</tr>
<tr>
<td>ST</td>
<td>−16 (−17; −14)</td>
<td>−11 (−15; −13)</td>
<td>0.06</td>
</tr>
</tbody>
</table>

a 100 ms before landing. 
 b 50 ms after landing.
displayed equal isometric knee flexion strength compared with controls, but with reduced knee flexor muscle activity (Jensen et al., 2013). However, direct comparisons should be made with caution due to different test situations, i.e. isometric knee flexion versus the current dynamic performance test, with gravity causing most of the knee flexion.

Also, it is possible that a higher tendon slack length in the GJH group may require higher GM muscle activity and force transfer to attain the same performance output or jump length as in controls. Reduced tendon stiffness, possibly due to structural changes, was observed in women with the connective tissue disease, Ehlers-Danlos syndrome (hypermobility type) (Rombaut et al., 2012). To our knowledge, no studies have evaluated tendon stiffness in GJH. Hypothetically, GM may produce a compensatory force output for the GJH group before landing, and thereby provide stability to the joint and/or resist anterior tibial displacement (Huston and Wojtys, 1996; DeMont and Lephart, 2004). The current higher GM activity was registered before landing, plausibly to compensate for the lower ST activity before landing as part of an anticipatory strategy. Remarkably, a corresponding compensatory higher GM activity after landing was not registered currently. This could be due to high anterior shear forces during impact (Griffin et al., 2006), plausibly hindering a higher activity in GM to be sustained.

The agonist function of the hamstring muscles to ACL, controlling Anterior Tibial Translation (ATT) is well documented (Griffin et al., 2006). Still, the function of the Gastrocnemii on the knee joint in weight-bearing, high-load functional tasks, such as landing from a single leg jump, is inconclusive. In vitro studies indicate the Gastrocnemii muscles to act as antagonist to the ACL, as Gastrocnemii force causes Posterior Femoral Translation (PFT) relative to the tibia and hence strain the ACL (Fleming et al., 2001; Elias et al., 2003). Conversely, in vivo studies suggested increased Gastrocnemii forces to compensate for decreased hamstrings forces during the weight-acceptance phase of single leg jump landing (Morgan et al., 2014), indicating a compensatory or agonist function to the ACL, similar to findings and hypothesis of the current study. Still, the effect of the Gastrocnemii on the PFT is questioned, as a low moment of force of the Gastrocnemii was found at peak ground reaction force during single leg jump landing in male adults (Podraza and White, 2010; Mokhtarzadeh et al., 2013). With large Gastrocnemii forces present, the knee joint reaction force will increase, which potentially could increase anteriorly directed tibia forces and consequently ACL loading (Mokhtarzadeh et al., 2013), thus the effect of the higher GM activity for the GJH group in relation to knee injuries need to be further studied.

The current lack of compensatory strategy for knee stabilization after landing may be one of the contributing factors for an increased risk of knee injuries in individuals with hypermobility and/or knee laxity, as previously reported in several studies (Ostenberg and Roos, 2000; Uhoroeh et al., 2003; Ramesh et al., 2005; Myer et al., 2008; Pacey et al., 2010). The reason may be that lower ST pre-activation implies a lower ST contraction force during impact or on initial part of the ground contact (Bencke and Zebis, 2011), thereby increasing the load on the ACL and the risk of injury for individuals with GJH. One study has demonstrated that non-contact ACL injury situations range from 17 to 50 ms after initial ground contact (Krosshaug et al., 2007), leaving no time for reflective or voluntary corrective muscular activation to prevent injury, indicating the importance of adequate pre-activation programming (Hewett et al., 2005).

Pre-activation programming, learned through previous experience to enhance correct timing and sequence of muscle contractions (Jones and Watt, 1971), could also be affected by disturbance of the proprioceptive system, which has previously been found in children (Fatoye et al., 2009) and adults (Hall et al., 1995) with symptomatic GJH. Reduced or disturbed proprioceptive feedback could potentially result in an altered ability to adjust neuromuscular patterns during landing. However, proprioception has never been tested in individuals with non-symptomatic GJH, and therefore the presence of proprioceptive disturbance in relation to non-symptomatic GJH is unknown.

The low ST activation levels for the GJH group could also be due to altered kinematics, such as decreased hip or knee flexion during landing. Landing with more extended knees could cause higher quadriceps activity for the GJH group, but this was not found in the current study. Also, the higher level of activity in the GM for the GJH group rejects this hypothesis, as GM is both a knee flexor and a knee joint stabiliser (Klyne et al., 2012). It is well known, that activation of the knee flexors may counteract the anteriorly directed shear force (Aagaard et al., 2000; Hewett et al., 2005), providing stability to the knee joint. Still, the role of the Gastrocnemius as an antagonist muscle providing knee stabilization seems to be minimal (Kellis, 1998).

Another important finding was the 39% higher CCI before landing for the lateral muscle pair, VL–GL, observed in the GJH group. A higher CCI for the lateral knee muscles may place increased stress on the medial side of the knee joint and the passive ligament structures, which again may lead to strain or rupture of ligaments. The higher lateral CCI, along with the lower ST activity and higher GM activity before landing might be a way to distribute compression forces of the joint equally, to control sagittal and horizontal movements and to provide the limb with dynamic stability (Aagaard et al., 2000; Solomonow and Krosgaard, 2001; Hewett et al., 2005). The CCI assessed for a functional task in the current study differs from co-activation ratios measured by isometric or isokinetic strength tests (Aagaard et al., 2000; Hewett et al., 2005; Jensen et al., 2013), although co-activation of the knee joint also has been assessed during functional tasks like jumping (Kellis et al., 2003; Masci et al., 2010; Podraza and White, 2010), applying an equation for co-activation by Winther (1990). This equation differs from the CCI, why direct comparisons to the current study are difficult. However, a functional CCI calls for careful interpretation, as EMG amplitude is affected by the type of muscle action, velocity of movement, level of effort and angular position (Kellis, 1998). Thus, the variation in results of the studies could be due to different set-ups or methods used for calculating the CCI. Even though the activation pattern was altered in the current study, no group differences were found in the isometric strength ratios for F/K/E or P/E/K/E between GJH and controls, similar to the findings of Jensen et al. (2013).

There are some potential limitations of the current study: (1) Mainly girls were included, which may hamper generalizability. However, since the prevalence of GJH among girls and women is higher in general, the current group may be well representative.
of the GJH group (Remvig et al., 2007). (2) Although the children were matched with respect to sex and age, they were not matched on sport type, where frequent participation in a specific sport may influence development of particular neuromuscular activation patterns. The influence of specific sporting activities on the current data set is therefore unknown. (3) A CCI was applied for analyses of co-contracting muscle work of the knee joint, while other studies have assessed the co-activation ratios of the knee joint muscles. Co-activation ratio is used when agonist and antagonist can be clearly defined over time in a specific task, while CCI can be used even though it is not possible to clearly define which muscles over time are agonist and antagonist muscles, as in the current task (single leg hop for distance) (Rudolph et al., 2001). Using the present calculation for CCI is feasible, since it gives the opportunity to compare the results with previous studies of similar populations where the same method of calculating CCI has been used (Greenwood et al., 2011a; b; Juul-Kristensen et al., 2014).

Since both mono-articular as well as bi-articular muscles are working in close cooperation to move and stabilize the knee joint, this presents a challenge for the interpretation. Given, that in the functional task studied it was not possible to fix the ankle/hip joint of the bi-articular muscles and isolate the effect to be mono-articular, both muscle groups were included for the CCI calculation, as similar to other studies of knee muscle co-activation (Masci et al., 2010; Podraza and White, 2010; Morgan et al., 2014). It is a weakness of the current study, that the bi-articular Rectus Femoris muscle is not measured, which was due to limitations of the available EMG channels. As the current primary aim was to study possible differences between GJH and controls in total knee joint loading, and the analyses are performed in a uniform way for both GJH and controls, we have no reason to suspect a bias in the results for GJH.

In conclusion, children with GJH and controls performed equally well in jump length. However, children with GJH had a GM-dominated neuromuscular strategy before landing, plausibly caused by reduced ST activity. Also, reduced ST activity was seen in GJH after landing, but with no compensatory GM activity to attain knee joint stability. Reduced pre and post-activation of the ST may present an important risk factor for traumatic knee injuries as ACL ruptures in GJH with knee hypermobility.

Conflict of interests

The authors declare they have no competing interests.

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