The Dose-Response Effects of Dissociation Training on Measures of Neuromuscular Control during Performance Screening in Male Youth Footballers

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i. Abstract

AIMS: Movement screens purportedly identify compensatory kinematics that predispose athletes to injury (Kiesel et al., 2011). The efficacy of assessing select competencies and prescribing remedial training based on screen outcomes however remains equivocal. The Foundation Performance Matrix Screen© (FPMS) supposedly profiles injury risk, subsequently directing its independent motor control Dissociation Training (DT) (Mottram and Comerford, 2008). However, there appears to be no research evidencing that DT can improve FPMS score or reduce injury. Therefore this study aimed to investigate the dose-response of DT on kinematic and kinetic measures of neuromuscular control in male elite academy footballers.

METHOD: The dose-response to DT therefore remains to be established. With institutional ethics approval, elite U15/16 and U17/18 male academy footballers comprised group one (n = 6) (G1) and group two (n = 8) (G2) respectively. G1 performed DT 1x week while G2 performed DT 3x week over eight weeks. Centre of pressure (CoP) total, anterior-posterior (*X*) and medial-lateral (*Y*) displacements (cm), sway velocity ($cm{\cdot}s^{-1}$) and ellipse area (cm^2) were recorded from participants' non-dominant leg during a single leg stance test (SLST) and Y balance test™ (YBT). Force platform time to stabilisation (TTS), peak vertical ground reaction force (PVGRF) and loading rates were recorded from a 20cm bilateral drop jump landing (DJL). The FPMS and YBT were scored according to respective guidelines. All tests were performed barefoot. Cohen's *d* effect size (ES) was calculated from differences in means.

RESULTS: Small ES for G1 (ES -0.180; 95% CI,−1.94 - 0.60) and G2 (ES −0.136; 95% CI, −0.12 - 1.62) FPMS scores were observed. Large ES for DJL loading rates (ES -1.89, 95% CI, 0.046 - 0.079) and YBT normalised anterior reach (ES 1.416, 95% CI, 66.30 - 73.29) were observed for G1 compared to G2 where trivial (ES 0.072, 95% CI, 0.067 - 0.095) and moderate effects (ES 1.104, 95% CI, 66.84 - 72.90) respectively, were observed. The magnitude of change for G1 was consistently greater for all DJL and YBT measures. Furthermore, SLST performance for G1 improved for all CoP measures whereas G2 decreased. **CONCLUSION**: The measures used to assess neuromuscular function indicate eight weeks DT had meaningful effects on neuromuscular control, however, the magnitude of effects were greater for G1 than G2. As SLST, YBT and DJL indicated greater effects and are all proposed to predict injury, they could be a suitable surrogate marker for assessing the effects of DT. These findings also suggest that a lower dose of DT is sufficient provided training is individualised.

ii. Declaration Statement

I declare that the work in this thesis was carried out in accordance with the regulations of the University of Gloucestershire and is original except where indicated by specific reference in the text. No part of the thesis has been submitted as part of any other academic award. The thesis has not been presented to any other education institution in the United Kingdom or overseas.

Any views expressed in the thesis are those of the author and in no way represent those of the University.

Robert M Burge

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iii. Acknowledgements

The completion of this MSc by Research has been a challenging yet rewarding journey, and one that would have not been possible alone. I hope that the findings of my research can be disseminated and offer practical application when designing prospective injury prevention screening and training prescription for youth athletes.

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Contents

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1.0 Introduction

Understanding the physiological match demands and injury epidemiology of a sport is central to designing an effective training programme (Russell et al., 2013, Di Salvo et al., 2007, Da Silva et al., 2007). While these parameters in elite adult footballers has been well documented (Bengtsson et al., 2013, Lundblad et al., 2013, Russell et al., 2013, Andrzejewski et al., 2012, Mendiguchia et al., 2012, Ekstrand et al., 2011, Hagglund et al., 2009, Werner et al., 2009, Di Salvo et al., 2007, Bangsbo et al., 2006, Stolen et al., 2005, Edwards et al., 2003), few studies have analysed elite academy youth footballers across a range of age groups (defined for the purposes of this review of literature as players representing professional club's academies, national or international centres of excellence, and international teams) (Deehan et al., 2007, Le Gall et al., 2007, Le Gall et al., 2006, Merron et al., 2006, Emery et al., 2005, Price et al., 2004, Kakavelakis et al., 2003, Volpi et al., 2003, Peterson et al., 2000). Elite adult players reportedly cover ~12km per game (Stolen et al., 2005) and perform up to 250 high-intensity match actions (such as rapid accelerations and decelerations, short sprints, cutting and change of direction (COD), bursts of unilateral and bilateral jumping and landing, ball striking and tackling) every 4 to 6s, totalling 1000 to 1400 per game (Di Salvo et al., 2007, Edwards et al., 2003, Young et al., 2001). Players also reach and maintain high-intensity running thresholds for

ranges of 3m to 40m (Bangsbo et al., 1991), on average, every 70s (Stolen et al., 2005). By comparison there is a greater discrepancy between average match distances covered and high-intensity match actions performed throughout elite academy age groups (Rebelo et al., 2014, Lago-Penas et al., 2011, Harley et al., 2010). U12 and U18 have reportedly covered total match distances of 6.2km and 9km respectively of which 9.2% comprised high-intensity match actions for both age groups (Lago-Penas et al., 2011). Whereas, U17 reportedly only covered 5km to 7km but ~15% represented high-intensity match actions (Rebelo et al., 2014). A corresponding trend between increased match exposure and absolute distance covered (r^2 = 0.542, P = 0.001) has also been observed in older age groups (Harley et al., 2010). Such disparities have been attributed to increases in pitch size, match duration and match frequency in older age groups (Russell et al., 2013). Although, GPS running thresholds corrected to age-velocity characteristics (relative to $m \cdot min^{-1}$) have indicated no significant differences ($P <$ 0.05) between U12 to U16 for match distance covered at high- (30.4% [17.1% - 42.6%]), very high- (11.9% [4.5% - 22.7%]) and maximal-intensity (3.6% [0.3% - 8.8%]) (Harley et al., 2010). These findings suggest that irrespective of pitch size and match duration elite academy footballers' work-intensity profiles are comparable throughout all age groups. This is of particular importance as running, cutting and COD have consistently been reported as the predominant cause of injury in academy footballers (Cloke et al., 2012, Mendiguchia et al., 2012, Hagglund et al., 2009, Price et al., 2004, Woods et al., 2004, Hawkins and Fuller, 1999).

Another compounding factor attributed to differences between age groups are the inherent variances in physical performance due to growth and maturational changes (Buchheit et al., 2010b). The rapid changes to youth players' immature musculoskeletal system purportedly heighten their predisposition to overuse injuries (Mersmann et al., 2014). This has been proposed as a major factor why academy footballers have a higher non-contact vs. contact injury ratio and incidence of non-contact injuries compared to elite adult players (Mersmann et al., 2014, Johnson et al., 2009, Le Gall et al., 2007, Hawkins and Metheny, 2001). Non-contact injury to the lower extremity also accounts for over 90% of the total incidence in elite academy football (Ekstrand et al., 2011, Hagglund et al., 2009, Price et al., 2004). While growth and maturational changes are commensurate with the onset of and circa-pubescent periods, the timing and rate that these changes occur varies greatly (Beunen and Malina, 2008). The present English Football Association's (FA) long-term Elite Player Performance Plan (EPPP) however assumes that growth and maturation progress linearly with age groups. By failing to consider these factors the FA's EPPP approach (to increase training and match intensity and frequency, pitch size and level of competition by age group) therefore possibly exacerbates injury incidence in academy players (Rumpf and Cronin, 2012). As the impact of injury in academy footballers' can directly affect their physical development and skill acquisition, the possibility of future success may be lessened (Charness, 1985). A lack of empirical analysis on the physiological match demands and injury epidemiology in academy footballers however means it is inherently difficult to quantify specific motor tasks that can increase injury risk. This highlights the complexity of determining

non-contact injury risk factors as well as workloads for adaptation and recovery when designing injury prevention training programmes for academy players.

A popular method of quantifying injury risk and programming subsequent remedial training is to employ a battery of kinematic competency assessments (Cook et al., 2006). A myriad of performance measures can be employed to assess an athlete's performance level or athletic potential (Parchmann and McBride, 2011). Performance assessments however often only produce an outcome measure that is not sensitive enough to determine any compensatory kinematics (Chorba et al., 2010, Parchmann and McBride, 2011). Therefore, irrespective of the outcome measure's relative score, poor motor patterns could be being reinforced potentially heightening an athlete's predisposition to injury (Peate et al., 2007). Consequently, kinematic assessments of select physical competencies are commonly employed as predictors of injury (Kiesel et al., 2011, Mottram and Comerford, 2008, Cook et al., 2006, Plisky et al., 2006). The validity of screening select physical competencies as predictors of injury however remains equivocal (Wingfield et al., 2004, Chalmers, 2002a). Likewise, empirical findings are inconsistent as to whether movement control and/or strength training prescribed in accordance with screen outcomes can enhance successive screen performance (McCall et al., 2014, Kiesel et al., 2011, Filipa et al., 2010, Kiesel et al., 2008, Cook et al., 2006, Plisky et al., 2006). As poor execution of select kinematic competencies is considered symptomatic of reduced motor control, movement control based remedial training is often preferentially focussed on ahead of traditional strength training (Hibbs et al., 2008). This is based on a plethora of pathological evidence of injury indicating

impaired lumbo-pelvic hip complex (LPHC) function causes greater uncontrolled joint displacements (Verrelst et al., 2014, Wilkerson et al., 2012, Chuter and de Jonge, 2012, Willems et al., 2006, Willson et al., 2006, Willson et al., 2005, Niemuth et al., 2005, Leetun et al., 2004, Ireland et al., 2003). Furthermore, cognitive control has also been identified as a pathological factor of injury (Verrelst et al., 2014, Verrelst et al., 2013, Leetun et al., 2004, Ireland et al., 2003, Fredericson et al., 2000, Bendjaballah et al., 1997). As movement control develops in a core-to-extremity and head-to-progression (Cook et al., 2006), this evidence advocates the implementation of movement control training in academy footballers could be desirable. A contemporary evidenced based Youth Physical Development model has advocated integrative neuromuscular training should always be present, in some capacity, to develop foundational motor skills in youth athletes (Lloyd et al., 2012b). The reinforcement of biomotor proficiency has also been proposed to maximise sport specific kinematic development and lower injury disposition in youth populations (Granacher et al., 2011, Myer et al., 2011a, Myer et al., 2011b, Faigenbaum et al., 2009). Most integrative neuromuscular training protocols however are multifaceted. Although neural changes have been reported to occur as early as four weeks, the scope for adaptation in trained athletes is much smaller (Behm et al., 2002). Furthermore, the efficacy of independent movement control training on motor control in adolescent athletes remains equivocal (Wright et al., 2015, Filipa et al., 2010, Stanton et al., 2004). One such motor control training modality is dissociation training (DT) which is independent to and directed by the Foundation Performance Matrix Screen© (FPMS). Through correcting the FPMS identified uncontrolled movement, DT can purportedly accelerate identified assets

considered central to effective athletic performance and reduce injury risk (Mottram and Comerford, 2008, Comerford and Mottram, 2001b, Comerford and Mottram, 2001a). The concept of dissociated movement however has reportedly only been applied effectively in clinical settings during isolated muscle function (Tsao and Hodges, 2007, Jull et al., 2002, Sahrmann, 2002, Hides et al., 2001, O'sullivan, 2000, Hamilton and Richardson, 1998, Woolsey et al., 1988). The effectiveness of DT on lowering injury risk in an athletic youth environment therefore remains to be evidenced. Furthermore, owing to the FPMS novelty, there appears to be no studies that have investigated the efficacy of FPMS and its associated independent DT strategies. Therefore this study aimed to investigate: (1) the dose-response of eight weeks DT on FPMS performance; and (2) whether changes in FPMS are in line with other kinematic and objective kinetic measures of neuromuscular control in male elite academy footballers.

2.0 Literature Review

2.1 Injury Epidemiology for Elite Youth Academy Football Players

The immature musculoskeletal system of a youth footballer, notably circa-pubescent, can heighten the risk of injury (Mersmann et al., 2014). Few longitudinal or large sample population studies however have investigated injury prevalence in elite academy footballers. To the author's knowledge there is only one large scale investigation conducted in English elite academy footballers. As part of the FA's medical injury audit Price et al., (2004) recorded 4773 players, aged 9 to 19 years, from 38 different English academies over two consecutive seasons (1999/2000 and 2000/2001). Price et al., (2004) reported 3805 injuries of which 50.4% and 48.7% occurred during competition and training respectively (with 0.9% accounting during no activity). An earlier investigation over three seasons (1994/95 - 1996/97) by Hawkins and Fuller (1999) however indicated competition accounted for 66% of youth players' injury occurrence. Alternatively, Le Gall et al., (2006) reported an injury occurrence of 30.9% and 69.1% for competition and training respectively for elite French youth players over 10 seasons (1993/94 - 2003/04). Despite longer study durations, the greater injury ratios between competition and training reported could be attributed to Hawkins and Fuller (1999) only investigating four English academies and Le Gall et al., (2006) a cohort of 66 players across three age groups each season (U14 = 24, U15 = 22 and U16 = 20). However, irrespective of injury ratios, high injury incidence per-1000 hours remain to be reported in elite academy footballers (Table 2.2 and Table 2.3).

Price et al., (2004) reported the most prevalent mechanisms of non-contact injuries during competition and training respectively were caused by running (16% and 23%), other non-contact (6% and 9%) and twisting/ turning (6% and 8%). Correspondingly, Hawkins and Fuller (1999) also reported the primary mechanisms of non-contact injuries during competition and training as running (7% and 12%) and turning (3% and 19%). The remaining non-contact mechanisms reported comprised jumping, landing, and stretching or overuse, accounting for 8% and 9% (Price et al., 2004) and 12% and 8% (Hawkins and Fuller, 1999) of competition and training respectively. Alarmingly, the sum of non-contact injuries reported by Price et al., (2004) (36% and 49%) and Hawkins and Fuller (1999) (22% and 39%) for competition and training represent large percentages of the total (non-contact and contact combined) injuries reported. Hawkins and Fuller (1999) further calculated that mechanisms underpinning non-contact injuries accounted for 59% vs. 41% for contact injuries of the total reported. Comparably, Volpi et al., (2003) followed Milan's professional academy players aged 9 to 19 years over four seasons, reporting non-contact and contact injuries accounted for 63.8% vs. 36.2% respectively. Emery et al., (2005) also reported a greater non-contact injury incidence of 53.85% vs. 46.15% for contact in U14, U16 and U18 elite Albertan academy league players. However, Emery et al., (2005) only analysed a single 13 week season which possibly explains the lower non-contact:contact injury ratio reported. By comparison, an investigation over three seasons in nine elite male adult European football teams indicated non-contact injuries only accounted for 26% vs. 73% for contact injuries (Hagglund et al., 2009). This evidence reinforces the premise that youth players are more susceptible to non-contact injury (Mersmann et al., 2014, Xu et

al., 2009, Gamble, 2008, Hawkins and Metheny, 2001). It should be noted however that non-contact injuries reported during competition (23%) and training (43%) by Hagglund et al., (2009) were comparable to Price et al., (2004) findings.

Le Gall et al., (2006) and Emery et al., (2005) investigated injury risk according to age group. Le Gall et al., (2006) reported that U14 sustained significantly (P = 0.04) more injuries per 1000-hours during training compared to U15 and U16. Furthermore, although there was no significant difference between groups (P = 0.96) U14 had a greater injury rate than U15 and U16 players. However, U16 sustained the greatest number of injuries per 1000-hours during competition. No significant difference ($P > 0.05$) was found for the severity of injuries in U14, U15 and U16. Similarly, Emery et al., (2005) also reported that U14 players in the top two Albertan elite divisions had a significantly (P = 0.04) greater relative risk of 2.45-injuries per 1000-hours. Furthermore, U14 had a greater injury rate per 1000-hours (7.88 [95% CI 4.51 - 12.77]) than U16 and U18. Likewise, U16 (5.68 [95% CI 3.25 - 9.21]) compared to U18 (3.22 [95% CI 1.29 - 6.61]) had a greater injury rate as well as relative risk (U16 1.77 vs. U18 1.0 injuries per-1000 hours) respectively. However, like Le Gall et al., (2006), no significant differences between age groups were observed. Emery et al., (2005) also found that the elite U14 were at significantly greater risk of injury than players in Division 2 (P = 0.003) and lower divisions (P = 0.0003). No significant differences were observed for the same in U16 and U18.

The impact of injury in academy footballers can directly effect a player's physical development and skill acquisition, and therefore possibly future success (Charness, 1985). Price et al., (2004) classified injury severity by time missed (slight = 2 - 3 days; Minor = 4 - 7 days; Moderate = 1 - 4 weeks; Severe = >4 weeks). Of the injuries sustained during training and competition respectively, Price et al., (2004) found 25% and 24% were for minor, 46% and 48% for moderate as well as 18% and 20% for severe. These findings are commensurate with the findings of Hawkins and Fuller (1999), Le Gall et al., (2006), Hagglund et al., (2009) and Merron et al., (2006) (Table 2.1). Interestingly, although more injuries occurred during competition, Price et al., (2004) and Hawkins and Fuller (1999) representation of injury severity is proportionate to training. This suggests the mechanisms of injury during competition and training are similar, which may explain the prevalence of the location and nature of injuries reported in Table 2.2 and Table 2.3. This cannot be ascertained for Le Gall et al., (2006), Hagglund et al., (2009) and Merron et al., (2006) as only a combined competition-training injury severity was reported. Nonetheless, minor and moderate severity injuries accounted for the majority of the total recorded by Price et al., (2004), Hawkins and Fuller (1999), Le Gall et al., (2006) and Merron et al., (2006). Although Hagglund et al., (2009) was an exception to this the author postulates this is likely because only 36 injuries were reported in a single age group.

Table 2.1: Injury Severity Prevalence in Elite Academy Players

	Price et al., (2004)				Hawkins & Fuller (1999)				Le Gall et al., (2006)		Hagglund et al., (2009)		Merron et al., (2006)	
Age Groups	**U9 - U19**				**NR**				**U14, U15, U16**		**U17**		**U16 - U18**	
Exposure	**TRN**		**COMP**		**TRN**		**COMP**		**T/C NS**		**T/C NS**		**T/C NS**	
Injury Severity	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**
Slight (2-3 Days)	183	10	180	8	6	11	18	17	357	31	26	72.2	84	36
Minor 4-7 Days)	459	25	466	24	23	40	29	29	337	29.3	1	2.8		
Moderate 1-4 Weeks)	842	46	912	48	22	39	47	43	344	29.9	5	13.9	88	37
Severe (>4 Weeks)	330	18	393	20	6	11	15	14	114	9.9	4	11.1	64	27

Percentage totals may be subject to rounding errors associated with independent components; TRN = Training; COMP = Competition; T/C NS = Injuries sustained during Training & Competition Not Specified

Of the studies reported in Table 2.1, Table 2.2 and Table 2.3 only three reported time missed by a player per season and only one for games per injury. Price et al., (2004) found academy players missed on average 21.9 ± 33.63 days per season and 2.31 ± 3.66 games per injury. Hawkins and Fuller (1999) however reported that the average days missed per season were roughly one-week less for competition (15.2 days) and training (13.0 days), whereas Merron et al., (2006) reported roughly one-week more (28.7 days per season). This variance between studies however could be because smaller samples and/or longer study durations of three (Hawkins and Fuller, 1999) and four seasons (Merron et al., 2006) were used. Another consideration is that Merren et al., (2006) only followed one academy and reported a much greater percentage of severe injuries than the other comparative studies. Therefore, the greater average time missed due to injury could be a reflection of the clubs training and competition schedule as oppose to representative of all academy populations.

Reported injuries to the lower limb account for 71% to 90% (Table 2.2) (Deehan et al., 2007, Le Gall et al., 2006, Merron et al., 2006, Emery et al., 2005, Price et

al., 2004, Volpi et al., 2003, Peterson et al., 2000, Hawkins and Fuller, 1999). The three most prevalent injury sites reported are the thigh, ankle and knee, with only Emery et al., (2005) reporting the groin and Volpi et al., (2003) the pelvis fractionally above thigh injuries (Table 2.2). Price et al., (2004) further classified that the hamstrings (34%) and quadriceps (25%) predominated the reported thigh injuries. Moreover, Price et al., (2004) reported muscular strains to the quadriceps (35%), hamstrings (33%) and hip adductors (20%) predominated recurring injuries. Correspondingly, Cloke et al., (2012) reported that the quadriceps (39.1%) sustained the greatest number of injuries, followed by the hamstrings (33.8%) and adductors (23.2%) (when investigating thigh muscle injuries in U9 to U16 from forty-one premier English clubs). However, Cloke et al., (2012) furthered that the hamstrings (40.8%) sustained a substantially greater number of severe injuries, followed by the quadriceps (34.3%) and the adductors (20.7%). Similarly, Le Gall et al., (2006) also reported that a high proportion of thigh injuries were to the hamstrings (25.4%) and quadriceps (26.8%), but added that strains affecting the groin (28.3%) was predominant. Interestingly, although not significantly different ($P = 0.64$), Le Gall et al., (2006) also found that U14 were more susceptible than U15 and U16 to these prevalent injuries (72.1% vs. 71.2% vs. 69.5%). U14 also evidenced a two-fold increased incidence of tendinopathies. This could be reflective of the timing of the growth spurt synonymous circa-14 years of age in males (Beunen and Malina, 2008). Similarly, Emery et al., (2005) found that U14 were at significantly ($P = 0.01$) greater relative risk of sustaining a slight or minor injury than U16 and U18. Likewise, U16 were at greater ($P = 0.01$) relative risk than U18 for sustaining minor injuries. These findings however are contradicted by Deehan et al., (2007)

who found the relative risk of injury for U14 was ~80%, whereas for U15 and U16 it was >90%, despite an injury incidence 0.6 per-1000 hours per player for all age groups. Likewise, Peterson et al., (2000) observed that injury incidence for U14 to U16 was lower than that for U16 to U18 high-level players (6.0 vs. 6.6 per 1000-hours).

Muscular strains, ligamentous sprains and muscular contusions predominated injury diagnosis ranging from 55% to 88% of the total injuries reported (Table 2.3) (Deehan et al., 2007, Le Gall et al., 2006, Merron et al., 2006, Emery et al., 2005, Price et al., 2004, Peterson et al., 2000, Hawkins and Fuller, 1999). Volpi et al., (2003) was the only exception reporting over a two-fold incidence of osteochondritis and fractures compared to muscular strains. Although, Volpi et al., (2003) investigated players circa-puberty who would have been more susceptible to growth disorders (Malina et al., 2004). Nonetheless, Hawkins and Fuller (1999) suggested the large incidence observed could be resultant of inadequate recovery from training and competition, and/or poorly designed strength and conditioning training. Price et al., (2004) reported ligament damage accounted for 28% of knee injuries of which 85% were to the medial collateral ligament (MCL). MCL injuries also accounted for 20% of recurring ligamentous sprains (Price et al., 2004). Consistently, Volpi et al., (2003) found the knee was the highest risk site (41%) and reported a considerably greater incidence of ligamentous sprains (Table 2.2 and Table 2.3). Moreover, Volpi et al., (2003) observed that 38% of knee injuries were caused by non-contact (24%) and overuse (14%). These findings correspond with Price et al., (2004) and Hawkins and Fuller (1999) findings that running as well as twisting/ turning are the

predominant mechanisms of non-contact injuries. Price et al., (2004) also reported that ligament damage accounted for 72% of all ankle injuries, with the anterior talofibular ligament (ATFL) representing 83% of all reported and 78% of recurring injuries. Similarly, Le Gall et al., (2006) reported that the majority of muscular strains were to the ankle (55.7%). These findings are also commensurate with other studies in table 2.2 that show the ankle, muscular strains and ligamentous sprains as a prevalent injury site and diagnosis respectively. However, unlike the aforementioned studies Peterson et al., (2000) recorded the nature of each injury to the prevalent lower extremity injury sites. Alarmingly, Peterson et al., (2000) reported that 50% for the groin, 19% for the thigh, 24% for the knee, 4% for the ankle and 41% for other lower leg sites was caused by overuse injuries. In addition, Peterson et al., (2000) reported that 90% for the groin, 54% for the thigh, 56% for the knee, 51% for the ankle and 57% for other lower leg sites accounted for non-contact injury. These findings are synonymous with the prevalent injury locations as well as the high-incidence of non-contact and recurring injuries reported by the aforementioned studies (Table 2.2). As Peterson et al., (2000) was the only investigation to report the nature of injury for independent sites, the author therefore postulates that these findings are likely representative of elite academy players aged U14 to U18. Of the total injuries sustained by academy players Price et al., (2004) found that 54%, with Hawkins and Fuller (1999) reporting a significantly greater ($P < 0.01$) 52.3%, were to the dominant side.

Table 2.2: Prevalent Injury Sites in Elite Academy Footballers

	Price et al., (2004)				Hawkins and Fuller (1999)				Le Gall et al., (2006)		Volpi et al., (2003)		Emery et al., (2005)		Deehan et al., (2007)		Merron et al., (2006)		Peterson et al., (2000)		Kakavelakis et al., (2003)	
Age Groups	**U9 - U19**				**NS**				**U14, U15 & U16**		**U9-U19**		**U14-U18**		**U9-U18**		**U16 - U18**		**U14 - U18**		**U12 - U15**	
Exposure	**TRN**		**COMP**		**TRN**		**COMP**		**T/C NS**		**T/C NS**		**T/C NS**		**T/C NS**		**T/C NS**		**T/C NS**		**T/C NS**	
Injury Site	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**IR**	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**
Thigh	344	19	383	20	16	28	23	21	282	24.5	7	9.7	2	0.28	211	34	27	12	81	14.5	19	9
Ankle	347	19	380	19	10	18	18	17	205	17.8	11	15.2	9	1.28	123	19.8	46	19	114	20.4	60	29
Knee	306	17	358	18	6	11	11	10	176	15.3	30	41	4	0.57	102	16.2	46	19	99	17.7	75	36
Lower Leg	183	10	192	10	5	9	10	9	60	5.2	2	2.7	5	0.71	-	-	17	7	53	9.5	13	6
Groin	179	10	172	9	9	16	11	10	-	-	-	-	3	0.43	44	7.0	15	6	41	7.3	-	-
Foot	156	9	128	7	3	5	10	9	95	8.2	4	5.5	2	0.28	42	6.8	20	8	56	10	-	-
Spine	81	5	81	4	-	-	-	-	-	-	-	-	2	0.28	42	6.8	17	7	33	5.9	-	-
Hip / Pelvis	63	4	88	5	1	2	5	5	25	2.2	8	11.1	-	-	-	-	19	8	-	-	11	5
Abdomen	12	1	14	1	-	-	-	-	5	0.4	-	-	-	-	-	-	14	6	-	-		
Upper Arm & Shoulder	21	1	14	1	-	-	-	-	21	1.8	3	4.1	-	-	27	4.3	-	-	50	9	25	12
Hand	28	1	9	0	-	-	-	-	69	6	2	2.7	-	-			-	-			-	-
Skull	11	1	26	1	2	4	12	11	16	1.4	1	1.38	3	0.43	-	-	3	1	20	3.6	6	3
Other	69	6	79	3	-	-	-	-	-	-	2	2.8	1	0.14	30	4.8	12	6	31	5.6	-	-

Percentage totals may be subject to rounding errors; TRN = training; COMP = competition; TCNS = training & competition injuries not specified; IR = incidence rate

Table 2.3: Prevalent Types of Injuries in Elite Academy Footballers

	Price et al., (2004)				Hawkins and Fuller (1999)				Le Gall et al., (2006)		Volpi et al., (2003)		Emery et al., (2005)		Deehan et al., (2007)		Merron et al., (2006)		Peterson et al., (2000)		Kakavelakis et al., (2003)	
Age Groups	**U9 - U19**				**NS**				**U14, U15 & U16**		**U9-U19**		**U14-U18**		**U9-U18**		**U16 - U18**		**U14 - U18**		**U12 - U15**	
Exposure	**TRN**		**COMP**		**TRN**		**COMP**		**T/C NS**		**T/C NS**		**T/C NS**		**T/C NS**		**T/C NS**		**T/C NS**		**T/C NS**	
Nature of Injury	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**IR**	**No.**	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**
Muscular Strain	583	33	558	29	30	53	30	28	176	15.3	7	9.7	10	1.42	121	18	-	-	183	33	49	23
Ligamentous Sprain	359	20	389	21	11	19	22	20	192	16.7	23	31.9	10	1.42	252	37	-	-	299	54	69	33
Muscular Contusion	88	5	211	11	9	16	35	32	352	30.6	-	-	-	-	40	5.8	-	-	23	4.2	43	21
Tissue Bruising	98	6	163	9	-	-	-	-	-	-	-	-	-	-	23	3.3	-	-	-	-	-	-
Tendonitis/opathy	89	5	73	4	-	-	-	-	108	9.4	4	5.6	-	-	18	2.6	-	-	-	-	15	7
Fracture	68	4	77	4	1	2	6	6	68	5.9	16	22.2	-	-	-	-	-	-	17	3.1	16	8
*Joint & Bone Disorders	207	10	130	6	-	-	-	-	72	6.3	16	22.2	-	-	-	-	-	-	-	-	-	-
Meniscal Tear	20	1	28	2	-	-	-	-	25	2.2	-	-	-	-	-	-	-	-	-	-	-	-
Capsular Tear	15	1	17	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Cut	8	0	23	1	0	0	5	5	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Other Overuse	9	1	14	1	1	2	1	1	19	1.6	-	-	-	-	-	-	-	-	-	-	-	-
Dislocation	14	1	7	0	-	-	-	-	10	0.9	-	-	-	-	-	-	-	-	14	2.5	6	3
Ligament Rupture	8	0	11	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2	1
Other Diagnosis	223	13	216	10	5	9	10	9	52	4.5	6	8.3	3	0.43	18	25.5	-	-	18	3.2	7	3

Percentage totals may be subject to rounding errors; TRN = training; COMP = competition; TCNS = training & competition injuries not specified; IR = incidence rate

**Joint & Bone Disorders includes Osgood-Schlatter's, Periostitis, Inflammatory Synovitis, Sever's Disease & Osteochondritis*

2.2: The Impact of Growth and Maturation on Injury and Neuromuscular Control

Le Gall et al., (2007) advocated that maturational status of players within chronological age groups should also be considered as an injury risk factor. As differences in biological maturity can effect physical performance (Buchheit et al., 2010b) this can potentially create an unfair physical- and athletic-advantage during training and competition (Meylan et al., 2010, Malina et al., 2004). This could have implications for both contact and non-contact injuries (Malina et al., 2000). This may also, in part, explain why Cloke et al., (2012), Price et al., (2004), Malina et al., (2004), Volpi et al., (2003) and Schmikli and Bol (1995) have all observed concomitant increases in injury incidence with age group, with a steeper rise from >14 years. For instance, male footballers aged 14 to 17 years with a tall stature of >165cm and weak grip-strength of <25kg had a greater prevalence of sustaining select musculoskeletal injuries (Backous et al., 1988). This relationship advocates somatic growth and maturational factors effecting strength development are precursors to injury. This study however should not be directly compared with the aforementioned as elite academy players were not used. Therefore the sample population are not likely to of had comparable training statuses.

Le Gall et al., (2007) investigated U14 at the French centre of excellence over ten seasons as the mean chronological age of each group was synonymous with age of peak height velocity (APHV) (13.3 ± 0.3 years; range: 12.3 - 14.4 years). Based on biological age, maturational status was defined as early if older than 1.0 year, normal if within 1.0 year and late if younger than 1.0 year of chronological age. Although Le

Gall et al., (2007) found no significant differences ($P < 0.05$) between maturational groups for overall injury incidence, early and normal maturers had a higher incidence of moderate injuries than late maturers. Moreover, early maturers had a significantly higher incidence of severe injuries resulting in an $\geq$4 week absence from training and competition. This could be reflective of the greater rate of the growth spurt and maturational changes observed in early maturers which would intensify the risk of injury (Beunen and Malina, 2008, Gasser et al., 2001, Hägg and Taranger, 1991). The extent of non-contact and contact injuries however was not reported. In support, Johnson et al., (2009) followed 292 players aged 9 to 16 years from Manchester United's academy over six seasons (2001-07) evidencing comparable findings to Le Gall et al., (2007). Johnson et al., (2009) analysis of covariance (ANCOVA) indicated no significant differences between maturational status and injury incidence after adjusting for competition and training exposure, stature and position played ($F = 0.3$, $P = 0.73$). Johnson et al., (2009) general log linear analysis did conclude however that maturational status, as well as competition and training exposure, explained 47% of the variance in injury incidence. Both investigations therefore advocate that players of differing maturational statuses can train and compete alongside one another. However, while Johnson et al., (2009) analysis confirms that maturational status and playing exposure is a risk factor, Le Gall et al., (2007) advocates that early and late maturers could be at a greater risk to certain mechanisms injury.

The English FA implements a long-term Elite Player Performance Plan (EPPP) whereby training and match intensity and frequency, match duration, pitch size, and level of competition all increase with age group. However, the EPPP's limited

assumption that growth and maturation progress linearly with chronological age fails to account for the associated intrinsic risk factors that heighten injury risk in youth footballers (Rumpf and Cronin, 2012). For instance, the immature musculoskeletal system undergoes architectural changes such as rapid increases in limb length and mass, subsequently creating greater moments of inertia (Hawkins and Metheny, 2001, Jensen and Nassas, 1988). For example, Hawkins and Methany (2001) found increases in limb mass and moment of inertia resulting from 4cm increased leg growth, in the absence of MTU hypertrophy, resulted in a 30% greater force requirement (of the thigh musculatures maximum pre growth spurt) to generate the same angular acceleration to kick a football. However, there is a delay in the structural development of musculo-tendon units (MTU) and ligaments creating a time lag whereby the tensile strength of connective tissue remains closer to their failure limits (Hawkins and Metheny, 2001). Most notably is the time delay in the growth of muscle length and cross-sectional area (Xu et al., 2009). This is important as mechanical and morphological changes to the MTU influence both athletic performance (Stafilidis and Arampatzis, 2007, Arampatzis et al., 2006) and injury predisposition (Couppé et al., 2013, Hansen et al., 2013, Arya and Kulig, 2010). For instance, the subsequent pre-load on the MTU increases, applying greater force through the tendons and apophyses (Mersmann et al., 2014). Consequently, this can lead to reduced flexibility and tissue stiffness around the joint, early onset of neuromuscular fatigue, and/or the manifestation of overuse and overload injuries such as patellar tendinopathies and apophysitis (as evidenced in Table 2.3) (Mersmann et al., 2014). As APHV is indicative of the onset of puberty, considered to occur between the ages of 13.8 and 14.2 years in males (Meylan et al., 2014, Philippaerts et al., 2006, Bayli and Hamilton, 2004), identifying APHV is critical for

programming the appropriate type and volume of training to enhance athletic potential and reduce the risk of injury.

Just like the musculoskeletal system, neurophysiological adaptations occur analogously during maturation. For instance, pre- and circa-pubescent youths have evidenced more reactive (closed-loop) feedback neural pathways, subsequently reducing neuromuscular efficiency (Lloyd et al., 2011b, Lazaridis et al., 2010, Oliver and Smith, 2010, Grosset et al., 2007, Croce et al., 2004, Lambertz et al., 2003). This type of monosynaptic inhibition is considered to be a protective mechanism for the immature musculoskeletal system (Lazaridis et al., 2010, Croce et al., 2004, Lambertz et al., 2003). Whereas feed-forward (open-loop) neural pathways and the structural development of the MTU, has been evidenced to improve in post-pubescent youths (Lloyd et al., 2011a). This advocates that natural alterations in neuromuscular function occur during maturation. Causations for the natural development of neural strategies during maturation have been attributed to faster muscle fibre twitch-times (Lin et al., 1997), increased intrafusal fibre development (Grosset et al., 2007) and decreased golgi tendon organ (GTO) size and number (Ovalie, 1987). All of which have a synchronous effect with the structural development of the immature musculoskeletal system (Gamble, 2008). For instance, maturity-related increases in androgen concentration levels would promote muscle fibre-type development and differentiation (Viru et al., 1999). Likewise, decreased GTO number and size are likely facilitated by an increase in MTU length and cross-sectional area (Xu et al., 2009, Ovalie, 1987). In addition, it is well documented that muscle strength increases during maturation (O'Brien et al., 2010). Yet it has also been consistently reported that changes in muscle mass and increased joint torque

(through increased limb lengths) do not account fully for enhanced force production (De Ste Croix et al., 2003, De Ste Croix et al., 2001). This therefore suggests alterations in neural circuitry.

Consideration of intrafusal fibres and GTO functions are of particular importance when selecting appropriate training modalities to enhance intermuscular coordination in adolescent athletes. This is because they operate in concert to maximise stabilisation and control across a joint by balancing the force output of opposing musculature (Zatsiorsky and Kraemer, 2006). Intrafusal fibres are specialised sensory afferent *y*-neurons, coiled around muscle spindles located in the body of skeletal muscle, which monitor the rate and extent of eccentric contractions (Chalmers, 2002b). These *y*-afferent neurons synapse with the muscle's Ia-efferent neurons within the spinal cord to initiate a myotatic reflex response, potentiating the ensuing contraction (de Villarreal et al., 2009, Jamurtas et al., 2000). Acting as an injury safeguard, GTO are also specialised neurons located near the MTU junction that monitor the tension generated by the agonist (Chalmers, 2002b). When the MTU is overloaded, GTO trigger Ib-afferent interneurons that synapse with the muscle's Ia-efferent neurons within the spinal cord (Moore, 2007). This inhibits the intrafusal fibres' impulses from the agonist by means of a feedback pathway (Gabriel et al., 2006). However, this inherent sensory feedback can limit the effectiveness to stiffen and stabilise a joint when tolerating high ground reaction forces (GRF) (Wilson and Flanagan, 2008) and joint shear forces (Lephart, 2000). Furthermore, injuries through poor agonist-antagonist muscle balance ratios have been reported (Jaric et al., 1995). This reciprocal inhibition therefore adheres to the premises that, firstly, skilled movements need to be pre-programmed (Verkhoshansky and Siff, 2009) and,

secondly, coordinated co-activation is controlled at a supra-spinal level through programmed sensory modulation of voluntary movement as opposed to reflex actions (Nielsen, 2004). This further highlights the importance of promoting feed-forward processes as part of an integrative neuromuscular training programme in adolescent youths (Myer et al., 2013, Lloyd et al., 2012a, Lloyd and Oliver, 2012, Myer et al., 2011b, Ford et al., 2008).

2.3 Kinetic Assessments of Neuromuscular Control

The maintenance of posture and balance equilibrium is controlled by the central nervous system (CNS) through visual, vestibular and somatosensory inputs (Ruhe et al., 2010, Matsuda et al., 2008, Paillard et al., 2006, Vuillerme et al., 2005, Fransson et al., 2004, Balasubramaniam and Wing, 2002, Bringoux et al., 2000). By measuring plantar centre of pressure (CoP) injury prevalence and adaptations to specific physical training can be quantified (Abdul Razak et al., 2012). This is because plantar CoP measures are proportional to: ankle torque generated by body segment displacement (Balasubramaniam and Wing, 2002, Winter, 1995); proprioceptive and feed-forward neural pathways each initiating different muscle synergies dependent on whether a task is static or dynamic (Baratto et al., 2002, Gatev et al., 1999); and the regulation of joint stiffness by the mechanical properties of the surrounding musculature (Balasubramaniam and Wing, 2002, Baratto et al., 2002). All contribute to the equilibrium of balance and maintenance of postural control. As the point of application of the global ground reaction force (GRF) vector around which the body sways, CoP can be quantitatively measured through anterior-

posterior and medial-lateral sway displacement, sway velocity and sway ellipse area (Takacs et al., 2014, Ruhe et al., 2010, Matsuda et al., 2008, Mochizuki et al., 2006, Kitabayashi et al., 2003, Masani et al., 2003). Any oscillations of the body's centre of mass (COM) outside of the CoP is often described as 'postural sway' (Mochizuki et al., 2006). Feed-forward and proprioceptive neural pathways purportedly modulate postural control and balance via anticipatory postural adjustments and compensatory postural adjustments respectively (dos Santos et al., 2014). Both respond to changes in the state of the body signalled by the magnitude of the COM's displacement and velocity (Masani et al., 2003). This velocity feedback is central to anticipatory postural adjustments owing to the inevitable electromechanical delay in neural activation from proprioceptive mechanisms (Voight et al., 1998).

It is during perturbed movements that challenge postural control that pathological mechanisms are often signalled by increases in CoP sway displacement (Knapp et al., 2011, Ruhe et al., 2010, Ross et al., 2009, Santos and Aruin, 2009). This premise holds true for both static and dynamic movements when maintaining COM within the base of support (BOS) in healthy individuals. Furthermore, individuals with superior coordination during simultaneous upper and lower body displacement have indicated reduced COM displacement and CoP sway (Stapley et al., 1999). Basnett et al., (2013) however suggested that individuals with chronic ankle instability could also demonstrate decreased CoP sway displacement during dynamic movements, postulating impaired sensory function around the ankle would limit the magnitude of displacement achievable. Basnett et al., (2013) based this premise on a strong correlation between Y Balance Test™ (YBT) composite score of reach distances and ankle dorsi flexion range (r = .30, r^2 = .09, P = 0.02). Conflictingly however,

Hubbard et al., (2007b) also investigated the YBT evidencing a positive bivariate correlation between increased CoP sway velocity and sway area (r = 0.69, P = 0.001) for individuals with chronic ankle instability. In addition, Hubbard et al., (2007b) found correlations between hip extension strength and dorsi flexion peak torque (r = .43, P = 0.01) and hip abduction strength and CoP sway area (r = .49, P = 0.01). This is reinforced by the premises of dos Santos et al., (2014) and Kiers et al., (2012) that afferent pathways between the ankle and the LPHC are associated. Therefore all these considerations need to be accounted for when interpreting CoP measures.

Barone et al., (2011) and Matsuda et al., (2008) compared dominant and non-dominant kicking legs plantar CoP sway characteristics during a single leg stance test (SLST) in adult amateur footballers. Although not significantly different, both investigations evidenced decreased postural sway displacement (cm) and velocity ($cm \cdot s^{-1}$) on the non-dominant leg. However, Barone et al., (2011) premised that this was likely attributable to superior sensory feedback through visual input. However, Matsuda et al., (2008) further found that compared to swimming, basketball and non-athletes, footballers demonstrated significantly ($P < 0.05$) lower high-frequency anterior-posterior and medial-lateral sway displacement (cm) for both legs. Matsuda et al., (2008) postulated that irrespective of the SLST isometric nature, the greater size and mass of the torso over the narrowed BOS creates an inverted pendulum. Therefore, any oscillations outside of the BOS (which is also the CoP during unilateral stance) would result in fast accelerations of the COM. As reduced high-frequency sway is indicative of anticipatory postural adjustments neural control (Matsuda et al., 2008) whereas low-frequency sway is indicative of visual inputs

(Barone et al., 2011), Matsuda et al., (2008) hypothesised that footballers demonstrated both superior anticipatory postural adjustments and compensatory postural adjustments pathways. Paillard et al., (2006) investigated adult national and regional level footballers who participated in football training every day and twice a week respectively. All participants had a minimum of 10.0 ± 3.0 years playing experience and had been free from injury for six months. Using the SLST on player's non-dominant kicking leg, Paillard et al., (2006) found significant differences (P = 0.001) between groups for CoP ellipse area (cm^2) (national: 30.1 ± 10.8 cm^2 vs. regional: 49.1 ± 25.3 cm^2) (P = 0.01) and sway velocity ($cm \cdot s^{-1}$) (national: 1.67 ± 0.5 $cm \cdot s^{-1}$ vs. regional: 2.17 ± 0.5 $cm \cdot s^{-1}$). These findings infer that, firstly, more elite players demonstrate superior motor control and, secondly, this is likely due to a dose-response to training. Using the same criterions, Pau et al., (2014) investigated 21 male elite academy footballers from two Italian professional teams that trained for a minimum of six hours per week (age 14.5 ± 0.2 years, stature 164.5 ± 5.6 cm, body mass 56.8 ± 6.8 kg). CoP variables for dominant and non-dominant kicking legs (tested on separate days) were measured using an RS Footscan pressure mat pre and post a repeated sprint ability (RSA) protocol. Designed by Buchheit et al., (2010a) to simulate match induced fatigue in adolescent male footballers the RSA comprised 6 x 2 15m shuttle sprints with 20s passive recovery. Pau et al., (2014) found significant ($P < 0.05$) increases for CoP ellipse area (cm^2), maximum anterior-posterior displacements (cm) and sway velocities ($cm \cdot s^{-1}$). Medial-lateral sway velocity ($cm \cdot s^{-1}$) was the only exception when comparing baseline and fatigued values. No interaction however was found between the dominant and non-dominant kicking leg for rest and fatigued conditions. A fatigue-index from the RSA protocol and a fatigue/rest sway ratio was further calculated to investigate if a correlation

between the fatigue-index and sway-fatigue/rest-ratio existed. Pau et al., (2014) observed a significant positive Pearson's correlations for CoP total displacement (cm) (r = 0.631, P = 0.01), anterior-posterior sway velocity ($cm \cdot s^{-1}$) (r = 0.577, P = 0.01) and medial-lateral sway velocity ($cm \cdot s\text{-}^{1}$) (r = 0.529, P = 0.014) but for the non-dominant leg only. This therefore suggests the non-dominant leg fatigues at greater rate during simulated match play and that postural sway increases with neuromuscular fatigue. As Matsuda et al., (2008) and Barone et al., (2011) only compared adult amateur footballers with other amateur athletes and non-athletes, it remains unknown how much the level of footballing ability selects for players with superior balance and postural control. While Paillard et al., (2006) findings advocate elite footballers demonstrate superior motor control to maintain balance, supported by Matsuda et al., (2008) inverted pendulum concept, Pau et al., (2014) show both dominant and non-dominant legs fatigue at a similar rate.

Neuromuscular control measures derived from force plate data can also be employed to assess postural stability during the transition from a dynamic to a stationary state (Wikstrom et al., 2004). A common assessment is time to stabilisation (TTS) and associated peak vertical ground reaction forces (PVGRF) and loading rates during a landing from a jump or predetermined drop-height (Ebben et al., 2010). Traditionally TTS has been employed to progress plyometric exercise intensity as the stress placed on the MTC can be quantified by the objective ground contact time (GCT) and VGRF measures (Flanagan et al., 2008). However, TTS has also been used to investigate the effects of functional ankle instability (functional ankle instability) on balance (Ross et al., 2005) and comparing GRF attenuation between kicking and stance limbs (Ross et al., 2004). This is because anticipatory postural adjustments

modulate ankle extensor activation prior to stance phase which prevent the body from destabilising under high GRFs (dos Santos et al., 2014). However, chronic ankle instability can inhibit postural control because compensatory postural adjustments (indicative of proprioceptive pathways) affect afferent signals of the LPHC (dos Santos et al., 2014, Kiers et al., 2012). This was evidenced by Ross et al., (2005) who found a significant difference (P = 0.03) and a moderate effect (ES = 0.4) between TTS for adults with functional ankle stability (1.98 ± 0.81s) and functional ankle instability (1.45 ± 0.30s). In support, Rowley and Richards (2015) found that peak VGRF (P = 0.001, cohen's d ES = 0.80) and loading rates (P = 0.001, cohen's d ES = 0.81) significantly decreased with increases in plantar-flexion range of motion. This may explain in part why Ross et al., (2005) evidenced greater TTS in the functional ankle instability group. Similarly, Ross et al., (2004) compared TTS and PVGRF loading rates on 30 amateur adult footballers dominant and non-dominant ball-striking legs using a unilateral 36cm drop-jump landing (DJL). Despite the premises that the stance/ non-dominant leg often exhibits superior motor control (Kellis and Katis, 2007, Kellis et al., 2001), no significant differences (P < 0.05) were found between the dominant (TTS = 2.57 ± 1.02s, PVGRF loading rate = 0.09 ± 0.01s) and non-dominant (TTS = 2.65 ± 1.00s, PVGRF loading rate 0.08 ± 0.0s) legs. Ross et al., (2005) and Ross et al., (2004) findings however are limited as they only sampled VGRF at 180Hz and 400Hz respectively. Whereas a systematic review of literature by Niu et al., (2014) found that VGRF values were significantly less (P = 0.03) when sampled at <1000Hz than compared to at ≥1000Hz.

Force plate data sampled at 1000Hz in male and female adolescent athletes has also been employed successfully to assess LPHC control and knee torque moments (Myer

et al., 2011a, Hewett et al., 2005, Myer et al., 2005, Hewett et al., 1999, Hewett et al., 1996). Furthermore, owing to its objectivity and relative ease to execute, TTS has been advocated as a more practical measure of neuromuscular function than contemporary kinaesthetic assessments (Wikstrom et al., 2004). This is because GRFs have been associated with lower extremity injury (Hreljac et al., 2000, Mizrahi and Susak, 1982) for which joint range of motion and angular velocity under a given force will govern the amount of energy dissipated when landing (Fong et al., 2011, Kulig et al., 2011, Yeow et al., 2009, Yu et al., 2006). For instance, Hewett et al., (2006) found that circa- and post-pubertal male and female high school athletes with greater LPHC control decreased VGRF and knee valgus torque during 31cm DJL. Increased VGRF have also been correlated with knee abduction moments (r = 0.88, P = 0.001) in high school athletes (Hewett et al., 2005). Similarly, Hewett et al., (2005) previously observed a 20% difference in VGRF between athletes with a history of ACL injury vs. no injury. Meta-analysis testing has also evidenced that greater loading rates were associated with individuals that had sustained lower extremity stress fractures (Zadpoor and Nikooyan, 2011). This evidence indicates PVGRF loading rates are a pathological risk factor of lower extremity injury. Oliver et al., (2008) findings highlight the importance of the DJL assessment for youth footballers. Oliver et al., (2008) investigated bilateral 20cm DJL pre and post a 42-minute soccer-specific intermittent non-motorised treadmill fatiguing protocol (simulating the demands of one-half of match play) in amateur youth footballers aged 15.8 ± 0.4 years (stature 1.73 ± 0.06 m and body mass 59.8 ± 9.7 kg). While impact force (N) sampled at 1000Hz was the only significantly different (P < 0.05) GRF variable pre (2135 ± 369N) and post (2499 ± 422N) increases in PVGRF were still observed. This indicates why TTS has been advocated as an appropriate measure

in youth populations, notably during circa-pubescent periods (Barber-Westin et al., 2005), where postural stability is central to establishing solid foundational strength and reducing the risk of injury (Wikstrom et al., 2009).

2.4 Kinematic Assessments of Neuromuscular Control to Prescribe Training

Kinematic competency assessments are used to evaluate whole-body dynamic stability, identify asymmetries and/or kinematic deficits, monitor rehabilitation progress, and to provide a baseline to prescribe individualised remedial training from (Overmoyer and Reiser, 2013, Kiesel et al., 2011, Parchmann and McBride, 2011, Chorba et al., 2010, Filipa et al., 2010, Hale et al., 2007, Plisky et al., 2006, Gribble et al., 2004, Olmsted et al., 2002). However, select physical competencies are principally employed to predict injury as field performance assessments often only produce an outcome measure that is not sensitive to compensatory kinematics (Chorba et al., 2010, Parchmann and McBride, 2011). Therefore, irrespective of the outcome measure's relative score, poor motor patterns could be being reinforced potentially heightening injury risk (Peate et al., 2007). For instance, Mornieux et al., (2014) found under decreasing time constraints (850ms, 600ms and 500ms) lateral trunk flexion increased ~150ms prior to foot placement in adult amateur footballers performing pre-planned cutting manoeuvres. However, while increases in lateral trunk flexion facilitated task execution, a linear regression with increased knee abduction moment (r = 0.41, P = 0.009) was also evidenced (Mornieux et al., 2014). Unanticipated foot placement has been associated with high ankle and knee injury incidence (Verrelst et al., 2014, Wilkerson et al., 2012, Chuter and de Jonge, 2012,

Willems et al., 2006, Willson et al., 2006, Willson et al., 2005, Niemuth et al., 2005, Leetun et al., 2004, Ireland et al., 2003), which is also shown in table 2.2 as prevalent site of injury in academy footballers. While the validity of kinematic screening remains equivocal (Wingfield et al., 2004, Chalmers, 2002a), compensatory kinematics during select task execution are considered symptomatic of reduced motor control (Parchmann and McBride, 2011, Chorba et al., 2010, Mottram and Comerford, 2008, Peate et al., 2007, Cook et al., 2006). Hence why remedial based movement control training is often preferentially focused on ahead of traditional strength training (Hibbs et al., 2008).

Conceptually, a controlled movement is defined as stabilisation of the LPHC musculature under perturbations in order to control the body's COM displacement (Filipa et al., 2010, Mottram and Comerford, 2008, Zazulak et al., 2007, Hewett et al., 2006, Won and Hogan, 1995). As greater variability in COM displacement is associated with postural instability (van Emmerik and van Wegen, 2002, Blackburn et al., 2000), anticipatory postural adjustments and compensatory postural adjustments neural control can be predicted from the velocity feedback. However, the intensity, velocity and type of muscle contraction can all affect asymmetry and movement control (Overmoyer and Reiser, 2013). The majority of match actions such as running gait cycle, cutting and COD, ball dribbling and striking, tackling and bounding require unilateral stabilisation (Pau et al., 2014). During upright unilateral stance an individual's BOS decreases resulting in a shift in the COM to realign the body's weight over the BOS and CoP (Takacs et al., 2014). Foot placement of the stance leg therefore will govern the position of the CoP subsequently determining the direction that the COM can be accelerated in (Sadeghi et al., 2001, Patla et al.,

1999, Beckman and Buchanan, 1995). Furthermore, during foot placement of the stance leg proprioceptive afferent signals at the ankle modulate postural adjustments at the LPHC in response to COM displacements (Friel et al., 2006, Bullock-Saxton et al., 1994). By the same action, the LPHC can modulate changes at the ankle (Friel et al., 2006, Bullock-Saxton et al., 1994). For instance, increased excursion distances on the YBT, simulating a single leg squat motor pattern, reportedly represents superior control, strength, balance and active range of motion at the ankle, knee and LPHC (Munro and Herrington, 2010, Hale et al., 2007, Plisky et al., 2006, Gribble et al., 2004, Olmsted et al., 2002, Earl and Hertel, 2001). Increased hip abduction significantly ($P < 0.05$) correlates with hip extension strength ($r = .70$), as well as both additionally correlating with increased posterior-medial excursions ($r = .51$) and posterior-lateral ($r = .49$) excursions respectively (Hubbard et al., 2007b). Hip abduction strength also positively correlates with CoP sway area ($r = 0.49$) advocating increased LPHC strength assists both dynamic and static motor control (Hubbard et al., 2007b). Furthermore, independent reach distances have been evidenced to identify individuals with chronic ankle instability (Plisky et al., 2009, Hubbard et al., 2007a). Hubbard et al., (2007b) furthered that individuals with chronic ankle instability indicated positive correlations between increased CoP sway area and sway velocity ($r = 0.69$, $P = 0.001$) when performing the YBT. Plisky et al., (2009), Hubbard et al. (2007a) and Hubbard et al., (2007b) findings therefore reinforce the notion that LPHC weakness could be a pathological factor of chronic ankle instability and vice versa. Moreover, a three-fold increase in the risk of lower extremity injury was evidenced by Plisky et al., (2006) in 130 male high-school basketball players aged 14 to 18 years with a YBT composite score less than 94% of leg length. Asymmetrical anterior reach differences of $\geq$4cm between left and right

feet also indicated a two-and-half-fold increase in the likelihood of lower extremity injury.

The efficacy of using kinematic assessments to predict injury risk, monitor training progress and to prescribe training from in youth population remains to be elucidated. Filipa et al., (2010) investigated female footballers (EXP: 15.4 ± 1.5 vs. CON: 14.7 ± 0.8 years) participating in 16 sessions over eight weeks of Integrative Neuromuscular Control Training focussed on enhancing trunk stabilisation and lower extremity strength. The EXP Integrative Neuromuscular Control Training comprised stable and unstable training, proprioceptive landings, plyometrics, and traditional strength and power exercises. As the YBT was used as the pre and post assessment of motor control, Filipa et al., (2010) ensured that none of the exercises replicated YBT kinematics to reduce learning effects. The EXP YBT composite scores significantly improved on left (pre 96.9% ± 10.1% & post 103.4% ± 8.0%; $P = 0.04$) and right (pre 96.4% ± 11.7% & post 104.6% ± 6.1%; $P = 0.03$) legs. No significant changes however were observed for the CON left (pre 97.4% ± 7.2% & post 93.6% ± 5.0%; $P = 0.09$) or right (pre 95.7% ± 5.2% & post 94.4% ± 5.2%; $P = 0.15$) leg YBT composite score. Further analysis also indicated significant increases and large partial η^2 ES for EXP right ($P = 0.008$, η^2 ES 0.41) and left ($P = 0.04$, η^2 ES 0.27) legs posterior-lateral, as well as posterior-medial reach distances for the left leg ($P = 0.03$, η^2 ES 0.3). Similarly, Wright et al., (2015) matched 22 adolescents by Functional Movement Screen™ (FMS) composite score. The EXP (13.0 ± 0.8 years) participated in 4 x 30-minute sessions over four weeks. Training focussed on quality of fundamental movement skills comprising exercises comparable to the FMS assessments which were all directed by the FMS's advanced corrective exercise

manual (Cook, 2010). Whereas the CON (13.8 ± 0.8 years) only participated in generic multisport activities. Both groups had comparable exposure time to their respective training. However, within group changes were comparable with EXP and CON both indicating no smallest worthwhile change (= >0.2 ±*SD*) and only trivial effects (based on adjusted change scores, 90% confidence limits [CL]) for FMS composite score. These findings infer that the dose-response to short-term independent fundamental movement skills training was not sufficient to overcome the stimulus presented by competitive sports play. Another issue with screening in youth populations was evidenced by Lloyd et al., (2015). Previously, in adult athletes a correlation between low FMS-score and injury (r = .76, P = 0.02) as well as a score of ≥14 has been significantly (P = 0.04) associated with injury (Chorba et al., 2010). Investigating male academy footballers from UK professional clubs Lloyd et al., (2015) found that U16 were more mature than U13 and U11, as well as significantly (P < 0.05) outperformed them on the FMS (Table 2.3). However, when an ANCOVA was applied to maturation, no significant between group differences were observed indicating that maturational status influences screen outcome. This also indicates that Chorba et al., (2010) findings that a score ≥14 identifies high risk athletes should not be applied in youth populations. As the FMS tests inline lunge, active straight leg raise, and rotary stability are biomechanically comparable to the FPMS tests three, five, seven and nine (Table 2.5), the FPMS could also be affected by maturational status. However, due to the novelty of Lloyd et al., (2015) findings and the absence of any literature supporting the FPMS this can only be inferred.

Table 2.4: Lloyd et al., (2015) Participant Characteristics and Baseline Composite FMS Scores

Age Group	Chronological Age (Years)	APHV (Years)	Baseline FMS Composite Score
U11	11.2 ± 0.5	- 2.78 ± 0.4	12.0 ± 1.5*
U13	13.2 ± 0.2	- 1.44 ± 0.79	12.5 ± 3.0 *
U16	15.6 ± 0.7	+ 1.25 ± 0.41	16.0 ± 2.0

** Denotes significant (P < 0.05) difference to U16 baseline FMS composite score before ANCOVA applied to maturational status*

2.4.1 The Foundation Performance Matrix Screen©

The Foundation Performance Matrix Screen (FPMS) is a kinematic assessment that creates a performance profile determining performance assets and priority injury risk factors (Mottram and Comerford, 2008). This performance profile subsequently directs the prescription of the FPMS independent dissociation training (DT) strategies. Through correcting the FPMS identified uncontrolled movement, DT can purportedly accelerate identified assets considered central to effective athletic performance and reduce injury disposition (Mottram and Comerford, 2008). However, while this concept of dissociated movement has been applied effectively in clinical settings (Sahrmann, 2002, Hamilton and Richardson, 1998, Woolsey et al., 1988), the effectiveness of DT on lowering injury predisposition and enhancing athletic performance remains to be evidenced. Furthermore, owing to the FPMS novelty, to the author's knowledge there are no studies that have investigated the efficacy of FPMS and its associated DT strategies.

The FPMS assesses uncontrolled movement in frontal, sagittal and transverse planes permitting the detection of compensatory motor control synergies with regard to anatomical location and direction (Comerford, 2006). Comprising five low-threshold and five high-threshold tests the FPMS assesses slow-low and fast-high load motor control tasks to identify neural inhibition and weakness respectively (Table 2.5) (Mottram and Comerford, 2008). This is achieved by evaluating non-fatiguing alignment and coordination impairments through low-threshold testing, and dysfunction under fatiguing strength and speed during high-threshold tests (Mottram et al., 2014). All ten tests are multi-joint and assess specific kinetic chain dysfunctions at eight sites (upper neck, lower neck, upper back, shoulder blade, shoulder joint, low back/ pelvis, hip and lower leg) and in six directions (flexion, extension, rotation, side-bend, abduction and adduction). In addition, each test has six pass or fail questions regarding whether predetermined motor tasks can be controlled. Motor control dysfunction therefore can be attributed to specific sites, directions and loads (i.e., low- and high-threshold). This design purportedly allows practitioners to determine mechanical subgroups which may be the source of dysfunction more accurately, assisting the direction of subsequent movement control retraining strategies (Dankaerts et al., 2006, Sahrmann, 2002, Comerford and Mottram, 2001a).

Table 2.5: The Foundation Performance Matrix Screen Test Battery Order

Low Threshold Tests	
1.	Double Knee Swing
2.	Single Leg 1/4 Squat + Hip Turn
3.	Bridge + Straight Leg Lift & Lower
4.	Controlled Shoulder Internal Twist
5.	Four Point (Position) - Reach Forward & Back
High Threshold Tests	
6.	Supine (Position) - Single Straight Leg Heel Touch
7.	Plank + Lateral Twist
8.	One Arm Wall Push
9.	Split Squat + Fast Feet Change
10.	Lateral Stair Hop + Rotational Landing Control

The FPMS is grounded on evidence that synergistic dominance compensates when chronic and/or recurring musculoskeletal pain is caused through uncontrolled movement (O'Sullivan et al., 2006, Hodges and Moseley, 2003, Sterling et al., 2001). As the synergist primarily mobilises the joint the pattern of neuromuscular recruitment changes (Sahrmann, 2002). Therefore, neural synergies synonymous with high-threshold tasks (i.e., athletic performance) are recruited habitually to execute low-threshold non-fatiguing tasks (i.e., postural control); a task whereby joint stabilisers should be predominant (Ngomo et al., 2015, Dankaerts et al., 2006, Moseley and Hodges, 2006, O'Sullivan et al., 2006, Falla et al., 2004, Hodges, 2003, Hodges and Moseley, 2003, Comerford and Mottram, 2001b, Sterling et al., 2001). For example, Ngomo et al., (2015) found individuals' suffering from rotator cuff tendinopathy decreased infraspinatus excitability through cortico-spinal inhibition. SEMG RMS-amplitude values for the infraspinatus of the injured and uninjured shoulder were recorded preceding transcranial magnetic stimulation (TMS) pulses over the contralateral hemisphere to the shoulder tested. Ngomo et al., (2015) found significantly ($P = 0.01$) greater stimulation was required to induce an active motor threshold response in the infraspinatus of the injured shoulder. Furthermore, a

significant correlation (r = 0.45, P = 0.005) indicated concomitant increases between inter-hemispheric asymmetry of active motor threshold and the duration of chronic pain. Ngomo et al., (2015) concluded that chronic musculoskeletal pain suppresses infraspinatus excitability, advocating neuromuscular alterations occur at cortical-spinal level. The CNS changes observed by Ngomo et al., (2015) therefore advocate the implementation of motor control threshold testing and specific low-level neuromuscular training (such as the FPMS and DT) to identify and correct compensatory muscle synergies and reduce pain. However, their efficacy of enhancing neuromuscular performance and reducing injury in an athletic performance setting remain to be elucidated.

2.5: The Concept of Motor Control Re-Training

Motor control is the process by which the neuromuscular system coordinates muscle actions and limb movements to execute a given motor skill (Zatsiorsky and Kraemer, 2006). Within empirical literature this term is often used both interchangeably with and holistically to describe different training strategies such as movement control training (MCT), fundamental movement skills and Integrative Neuromuscular Control Training. However, the terminology used to describe specific motor control re-training strategies often cannot be operationally defined as they encompass the training of the same multiple physical factors in situ. For instance, Filipa et al., (2010) stated that the focus of Integrative Neuromuscular Control Training is to enhance *stabilisation* of the trunk and hip musculature and therefore control of the body's COM. This is because deficits in LPHC muscle synergies decrease trunk

proprioception, subsequently increasing COM displacement and the potential for biomechanical deviations (i.e., uncontrolled movement) in the lower extremity (Filipa et al., 2010, Mottram and Comerford, 2008, Zazulak et al., 2007, Hewett et al., 2006, Won and Hogan, 1995). Whereas, Won and Hogan (1995) proposed the definition of a *controlled* movement, the focus of MCT and fundamental movement skills, is one that it is *stabilised* against perturbations. It is therefore foreseeable why Mottram et al., (2014), Mottram and Comerford (2008) and Comerford and Mottram (2001a) classification of DT is comparable with other authors universal characterisation of motor control training.

Motor control develops in a core-to-extremity and head-to-toe progression (Cook et al., 2006) with pre-programmed integration of local, single- and multi-joint muscles activation resulting in proximal stability and distal mobility (Kibler et al., 2006). It is this proximal-to-distal patterning of force generation that creates moments that serve to protect distal joints (Borghuis et al., 2008). In support, deep segmental stabilising musculature activation has been evidenced to consistently precede limb movement by approximately 30ms to 100ms during dynamic whole body movements (Hodges and Richardson, 1997). It is the efficiency of this motor pattern to stabilise the LPHC that governs the production, transfer and control of force transmitted from the ground to the distal segments of the extremities (Hides et al., 2012, Kibler et al., 2006, Konin et al., 2003, Devlin, 2000, Hodges and Richardson, 1997, Hodges and Richardson, 1996). While neural control at spinal reflex and brain stem levels are predominantly governed by proprioceptive pathways (Radebold et al., 2001), autonomous intermuscular control can become learnt through repeated exposure (Kibler et al., 2006). It is these pre-programmed CNS controlled motor skills that

permit anticipatory postural adjustments muscle activations, controlling the body's COM displacement when perturbed by external forces (Kibler et al., 2006, Radebold et al., 2001). This was evidenced by Ebenbichler et al., (2001) and Kibler et al., (2006) who reported anticipatory postural adjustments increased proximal stability of LPHC allowing enhanced distal mobility as well as effective attenuation of GRF and loading of joints. Inhibition of LPHC stability through the onset of neuromuscular fatigue of abdominal muscles has also been directly associated with hamstring injuries (Devlin, 2000); the most prevalent non-contact injury in footballers. Thus, enhanced LPHC control has desirable implications for reducing predisposition to injury. Equally, enhanced LPHC motor control also has desirable implications for footballing performance as it aids the transfer of forces from the stance leg. Harrison (2006) and Barfield (1998) found concomitant increases in LPHC control and increased limb velocity in footballers' ball-striking leg. Any potential increase in the non-standing leg's angular velocity could reduce the muscle force required to overcome the inertia when executing football specific skills, as well as delay the onset of fatigue (Mersmann et al., 2014, Hawkins and Metheny, 2001). The potential greater impulse could also increase ball-striking velocity and therefore a player's passing and shooting distance (Barfield et al., 2002), as well as force output during tackling (Strauss et al., 2012). Both have been identified as determining factors of successful skill execution respectively (Strauss et al., 2012, Orloff et al., 2008).

While pathological evidence advocates impaired LPHC function causes the greatest uncontrolled joint displacements (Verrelst et al., 2014, Wilkerson et al., 2012, Chuter and de Jonge, 2012, Willems et al., 2006, Willson et al., 2006, Willson et al., 2005,

Niemuth et al., 2005, Leetun et al., 2004, Ireland et al., 2003), cognitive control of proximal mechanisms governing distal segment displacement has also been evidenced as a central pathological factor of injury (Verrelst et al., 2014, Leetun et al., 2004, Ireland et al., 2003, Bendjaballah et al., 1997, Fredericson et al., 2000, Verrelst et al., 2013). This evidence, as well as lower limb non-contact injury accounting for over 90% of the incidence in youth football (Ekstrand et al., 2011, Hagglund et al., 2009, Price et al., 2004), gives credence to the implementation of MCT and fundamental movement skills to enhance kinaesthetic awareness in youth players. Determining 'correct' motor patterns however is complex because instability, resulting in uncontrolled movement, increases co-contraction subsequently reducing force generating capacities (Anderson and Behm, 2005). Yet the ability to perform controlled movements cannot be achieved in the absence of coordinated synergist-antagonist intermuscular coordination stabilising the joint against perturbation and reducing displacement (Anderson and Behm, 2005, Lloyd, 2001, Kearney and Hunter, 1990). Lloyd (2001) summarised that enhanced coordinated synergist-antagonist intermuscular coordination should result in decreased electromechanical delay between perturbation and the onset of the resisting forces. Comerford and Mottram (2001b) therefore postulate that the onset and pattern of motor unit activation is central to the maintenance of balance and the efficiency of dynamic movements as oppose to force generation alone. This notion is why motor control training strategies focussed on movement competency are often preferentially recruited ahead of traditional strength training protocols for remedial training (Hibbs et al., 2008). However, it should be noted that integrative Integrative Neuromuscular Control Training interventions comprising resistance and plyometric exercise, traditionally considered high-threshold and dynamically correspondent

(Verkhoshansky and Siff, 2009), have consistently evidenced enhanced LPHC control and ability to attenuate GRFs (Myer et al., 2011a, Hewett et al., 2005, Myer et al., 2005, Hewett et al., 1999, Hewett et al., 1996). Whereas the efficacy of independent motor control training set from kinematic screening to reduce injury risk remains equivocal.

2.5.1: Dissociation Training

DT purportedly accelerates the identified performance assets and priority injury risk factors that comprise the FPMS performance profile (Mottram and Comerford, 2008). The respective differences between DT exercises for each have been outlined as: challenging movements that fast-track learnt skills with a low risk of injury; and low-threshold exercises that regain control of the site and, more distinctively, direction of the dysfunction (Mottram and Comerford, 2008). Comparable with other motor control strategies DT is aimed at enhancing neuromuscular coordination by integrating low- and high-threshold bodyweight exercises that challenge the kinetic chain's global stabilisation and local mobilisation (Mottram and Comerford, 2008, Comerford and Mottram, 2001a, Comerford and Mottram, 2001b). Characteristically DT involves the conscious isometric activation of global and/or local stabilising musculature (dependent on exercise threshold) to hold the site of dysfunction in a neutral position, while concurrently producing movement through an active range of motion at another (i.e., cognitively dissociating between joint stabilisation and mobilisation) (Mottram and Comerford, 2008, Comerford and Mottram, 2001b). This concept of dissociated movement however has reportedly only been applied

effectively in clinical settings during isolated muscle function (Tsao and Hodges, 2007, Jull et al., 2002, Sahrmann, 2002, Hides et al., 2001, O'sullivan, 2000, Hamilton and Richardson, 1998, Woolsey et al., 1988). Likewise, the studies highlighted by Mottram and Comerford (2008) that supposedly advocate that traditional strengthening (high-load and -velocity training) of the LPHC did not correct dysfunction or enhance motor control in the local stability system were tests of isolated muscle function only (Tsao and Hodges, 2007, Moseley and Hodges, 2006, O'Sullivan et al., 1997). For instance, Tsao and Hodges (2007) compared RMS SEMG activation of the Transverses Abdominis, internal and external Oblique, Rectus Abdominis, Erector Spinae, and anterior and posterior Deltoid following isolated motor control and sit-up training protocols to determine which had the greatest adaptation on anticipatory postural adjustments. No significant differences were found ($P = 0.74$) with the exception of Transverses Abdominis which indicated earlier onset of activation post intervention ($P = 0.001$). However, no significant difference ($P = 0.14$) was observed between the two protocols suggesting similar musculature recruitment strategies. Moreover, Umphred et al., (2001) and Janda (1993) have previously postulated that the Transverses Abdominis role during dynamic-athletic movements is resisting extension eccentrically, as oppose isolated-isometric or contracting concentrically during flexion. The effectiveness of DT on lowering injury predisposition and enhancing performance in an athletic environment therefore remains to be evidenced.

Correcting the direction of uncontrolled movement, identified by the FPMS, is purportedly what makes DT as a retraining strategy distinctive. By controlling the direction of dysfunction Comerford and Mottram (2001a) postulated the mechanical

stress at the site of provocation can be attenuated. Comerford and Mottram (2001a) furthered that this also allows the identification of tonic and phasic muscles involved, assisting the subsequent prescription of exercise thresholds. This follows an earlier concept of relative stiffness being direction-dependent (Sahrmann, 2002, Janda, 1993, Sahrmann, 1992). Sahrmann (2002) stipulated that if a muscle is too weak to adequately contract concentrically or resist eccentric loading, synergistic dominance occurs resulting in excessive motion at the joint of dysfunction. Therefore such compensations during athletic performance would be re-enforcing poor motor patterns (Peate et al., 2007, Comerford and Mottram, 2001b). This concept gives credence to Hewett et al., (2006) proposal that neuromuscular deficits in adolescent athletes leading to injury are muscle strength, expression of force, and/or pattern of activation that result in increased joint loading. Despite this, Comerford and Mottram (2008), Mottram and Comerford (2001a) and Mottram and Comerford (2001b) state the focus of DT is to alter the pattern and onset of activation, adding that strength is not a focus. Confusingly, Mottram and Comerford (2001a) also stipulate that DT should not be performed to- or under-fatigue to avoid compensation. Yet repetition ranges of 15-20 with active-end range of motion held isometrically for up to three-seconds have been advocated when performing low-threshold exercises (Comerford and Mottram, 2001a). Such repetition ranges and accumulative time-under-tension are commonly associated with training muscular endurance (Schoenfeld et al., 2015, Fry, 2004, Campos et al., 2002). Furthermore, this conflicts with Mottram and Comerford (2008) FPMS operational definitions of motor control and strength training (Table 2.6).

Table 2.6: Stabilisation retraining strategies: Operational differences between motor control and resistance-strength training as outlined by Mottram and Comerford (2008)

MOTOR CONTROL	STABILISATION STRENGTHENING
Muscle Specific: Training can be biased for either a local stability muscle role or a global stability muscle role depending on the cuing and facilitation used	**Muscle Non-Specific**: During high load resistance or endurance overload training to the point of fatigue all relevant synergists are significantly activated. There is co-contraction of the local stability muscle system, global stabiliser and global mobiliser muscle roles
Recruitment Specific: Because all these exercises use low load or functional normal loads then slow motor units are predominantly recruited	**Recruitment Non-Specific**: Again, because of overload, both slow and fast motor units are strongly recruited.
CNS Modulated: Afferent Spindle input influences CNS processes and tonic motor output	**Adaptation to Load and Demand**: Muscle Hypertrophy is a response to overload training

Comerford and Mottram (2001a) postulate that while DT is not reflective of athletic multi-segmental dynamic kinematics, DT exercises are fundamental movements that everyone should be able to perform competently. This notion corresponds with Lloyd et al., (2012) Youth Physical Development model which advocates that the development of foundational movement should always be present in some capacity in any type of youth strength and conditioning programme, with circa-puberty highlighted as key phase (Deli et al., 2006). This is because the mastery of foundational biomotor control is considered central for the successful development of complex sport-specific motor skills (Lloyd and Oliver, 2012, Lloyd et al., 2012b, Oliver et al., 2011). Multiple studies however have reported that the repetition of specific athletic kinematics and/or use of a dominant limb leads to natural synergistic and hypertrophic asymmetric adaptations (Hides et al., 2012, Hides et al., 2008, Ranson et al., 2008, Engstrom et al., 2007, Hides et al., 2007, Lucki and Nicolay, 2007, Kearns et al., 2001). For instance, Hides et al., (2012) and Kearns et al., (2001) reported different cross-sectional area and altered motor patterns for contralateral quadratus lumborum (QL) and ipsilateral psoas major muscles of professional

Australian Rules and junior English football players dominant legs. As both sports have comparable kinematic patterns these findings appear to support the premise of training dynamically corresponding movement for specific athletic motor control transfer (Verkhoshansky and Siff, 2009, Nielsen, 2004). This may also explain why Kellis et al., (2007) and Jackobsen et al., (2011) found that unilateral balance and postural control developed superiorly in trained youths and untrained adult footballers non-dominant stance legs. Furthermore, Danneels et al., (2001) reported that three weekly sessions over 10 weeks of low-threshold stabilisation DT failed to attenuate high-threshold dysfunction or multifidus atrophy. Whereas three weekly sessions over 10 weeks of combined low-threshold stabilisation DT and dynamic-static resistance strength training significantly increased multifidus cross-sectional area and enhanced motor control. This therefore supports the implementation of a multi-faceted programme when preparing athletes for high-intensity training and competition. The majority of research on DT however has been in clinical populations. Thus, it remains to be elucidated whether an independent DT programme can provide a sufficient stimulus to alter the synergistic dominant motor patterns associated in elite academy footballers. Furthermore, the dose-response required to induce the desired neural adaptations. While neural adaptations have been reported as early as four weeks, often attributed to initial physiologic enhancements in trunk strength and balance (Behm et al., 2002), the scope for adaptation is much smaller in trained populations (Cosio-Lima et al., 2003). Therefore this study aimed to investigate: (1) the dose-response of eight weeks DT on FPMS performance; and (2) whether changes in FPMS are in line with other kinematic and objective kinetic measures of neuromuscular control in male elite academy footballers.

3.0 Method

3.1 Participants

Eighteen elite male academy footballers volunteered for this study. Participant's characteristics are presented in table 3.1. One trial session was provided prior to the study to familiarise the participants with the testing equipment and the testing protocols. All participants had followed individualised DT programmes prior to the investigation. Participants were required to complete 80% of the total DT sessions, be available for pre and post testing, and be free from injury to be included for final analysis. Exclusion from the study was subject to these criterions. As a result of this stipulation four participants were removed from the study. Club consent was obtained and written informed consent was sought from each participant and participant's parent/ guardian. A health questionnaire was employed to ensure participant welfare before testing commenced. All participants were free from injury and illness. This study was approved by the University of Gloucestershire's institutional Research Ethics Sub-Committee.

Table 3.1: Participant Characteristics

Group	Participants	Chronological Age (Years)	APHV (Years)	Maturational Offset (Years)	Body Mass (kg)	Stature (cm)	Leg Length (cm)
G1	6	15.53 ± 0.47	15.31 ± 0.56	0.22 ± 0.54	65.55 ± 6.02	176.44 ± 4.06	90.17 ± 1.83
G2	8	17.36 ± 0.39*	15.89 ± 0.39	1.47 ± 0.44*	71.73 ± 6.79	177.51 ± 6.02	88.74 ± 4.49

**Denotes a significant difference between groups*

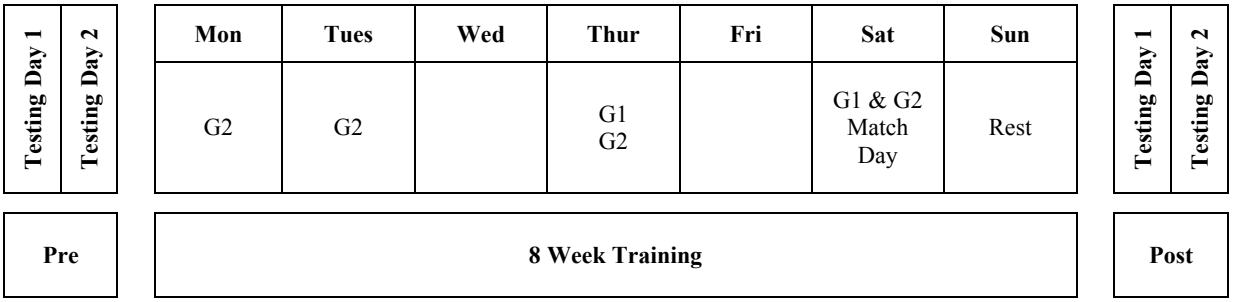
3.2 Training Intervention & Study Design

Participants were recruited from a professional football club academy representing the following chronological age groupings under (U) U15, U16, U17 and U18. All DT was prescribed by the football club's sport science staff in accordance with the FPMS outcome. The identified high risk areas of injury by the FPMS for the U17 and U18 however were predominantly the same causations for site and direction of dysfunction. Whereas, the high-risk injury areas identified for U15 and U16 were predominantly independent to the individual. As U17 and U18 performed DT three times per week and had comparable FPMS outcomes (subsequently directing comparable DT) they comprised G2 and performed a generic DT programme throughout the intervention. As U15 and U16 trained once per week and largely recorded independent FPMS outcomes they comprised G1 and continued with their individualised DT programmes.

The DT sessions were split into low and high threshold (intensity) motor control exercises (stipulated by the FPMS). As low intensity exercises always preceded high intensity exercises no warm up was prescribed. The investigator considered the increments in exercise intensity of each training session provided appropriate levels of mobilisation and activation for the target musculature. In addition, the FPMS does not advocate the requirement for a warm up prior to DT. The investigator therefore wanted to ensure continuity for the participants from pre-season training. All of G1 and G2 DT sessions were supervised by sport science staff, performed in the same building and at the same time in the morning prior to all technical training. Each DT session lasted 20 minutes. Subsequent training for G2 was not performed until 24

hours and 48 hours after the last completed session (Figure 3.1). DT volume and intensity was not changed during the intervention for G1 and G2. This was stipulated by the sport science staff in accordance with the participant's FPMS outcome. All participants were instructed to continue participating in all academy football training and physical education. Match frequency and total game time for each participant during the intervention and the season was recorded (Table 4.1).

Participants were tested pre and post the eight-week DT intervention. Testing was performed over consecutive days. The SLST, YBT and DJL were performed on testing day one. The FPMS was performed on testing day two. CoP measures were recorded for the non-dominant leg only. A schematic of G1 and G2 DT intervention and the study design is presented in figure 3.1.



Testing Day 1 = SLST, YBT and DJL

Testing Day 2 = FPMS

Figure 3.1: A Schematic of the study design and groups DT frequency

3.3 Testing Procedures

3.3.1 Anthropometric Measures

Participant's chronological age (years), date of birth (DD.MM.YYYY) and non-dominant leg (determined by asking the participant their preferred foot when striking a football (Peters, 1988, Chapman et al., 1987)) were recorded. Body mass (kg) was recorded using electronic scales (Seca 813 digital flat scales, Seca UK, Birmingham), stature (cm) using a portable stadiometer (Seca 217 stadiometer, Seca UK, Birmingham) and leg length (cm), measured in a supine position from the anterior superior iliac spine (ASIS) to the centre of the ipsilateral medial malleolus, using a tape measure (Seca 201 ergonomic circumference measuring tape, Seca UK, Birmingham). Predicted APHV was calculated using Mirwald et al., (2002) maturational offset equation.

*Maturity offset = ((-9.376 + 0.0001882) * (Leg Length and Sitting Height Interaction) + (0.0022 * Age and Leg Length Interaction) + (0.005841 * Age and Sitting Height Interaction)) – ((0.002658 * Age and Body Mass Interaction) + (0.07693 * Body Mass by Height Ratio))*

Age (Years), Leg Length (cm), Sitting Height (cm), Body Mass (kg)

3.3.2 Centre of Pressure

Transducers embedded in the Footscan pressure mat (High Speed 106.8cm x 41.8cm x 1.2cm Hi-End Footscan System, RSScan International, Olen, Belgium) recorded the anterior-posterior (F_y) and medial-lateral (F_x) plantar CoP displacements. For analysis plantar CoP measures were digitally normalised to participants' body mass (kg) and foot size (inches) and mean±*SD* sway velocity (cms $^{-1}$) calculated by the

Footscan balance software (Footscan Balance Version 7, RSScan International, Olen, Belgium). The displacement of CoP was calculated using equation 3.1 and equation 3.2. Plantar CoP measures were sampled at 15Hz for 60s for the SLST and 70Hz for the YBT. Sampling frequencies were limited by the Footscan balance software which predetermined the sampling frequency based on the input time to perform a task.

Equation 3.1 (Lafond et al., 2004)

$$CoP_x = ((-M_y + F_x * Z_0) / F_z) + X_0$$

Equation 3.2 (Lafond et al., 2004)

$$CoP_y = ((M_x + F_y * Z_0) / F_z) + Y_0$$

3.3.3 Vertical Ground Reaction Forces

VGRF were recorded using a 2-axis portable force platform (35cm x 35cm x 4.5 Pasco Force Paltform PS-2141, Pasco Scientific, Foothills Blvd, USA). Raw data was processed digitally using SparkVue software (Pasco SparkVue Version 2.0.52, Pasco Scientific, Foothills Blvd, USA). Before each testing session the unloaded force platform was left to stabilise with ambient room temperature for a minimum of 30 minutes to decrease potential electronic drift. VGRF data from the unloaded force platform was then sampled for 1s at 20Hz to record any transduced signals. A mean bias was calculated from these values (representing the mean shift of transduced signals) and removed from the experimental data to ensure that the force platform data had zero drift after amplification (Lafond et al., 2004). All force platform data was sampled at a frequency of 1000Hz. All recordings were manually triggered via the SparkVue software with the real-time data transmitted to the laptop computer via

a USB sensor connection (PasPort PS-2100A, Pasco Scientific, Foothills Blvd, USA). The investigator manually zeroed the force platform after each test.

3.3.4 Single Leg Stance Test

Participants stood barefoot unilaterally on their non-dominant leg aligned with a centre line and a toe marker to ensure consistent start positions on the pressure mat. The SLST was standardised by placing hands on hips, the non-standing leg was flexed to 90^{o} (the femur parallel to the floor, tibia perpendicular to the floor and ankle dorsi-flexed) and the torso and head upright maintaining a neutral spine. A demonstration was provided by the investigator for each participant. This static stance was required to be held for 60s. Participants received verbal instruction during the SLST to re-adopt the stance in the event they did not maintain the start position. All trials were performed with eyes open. Participants received three practice trials and three experimental trials to minimise any learning effect. Two minutes rest intervals were given between experimental trials. Participants SLST experimental trials were scored as a pass or a fail by the investigator. This was determined by correct execution of the SLST and whether all instructed criteria had been met. Participant's best SLST score from the three experimental trials was used for final analysis. CoP measures were recorded for each experimental trial.

3.3.5 Y Balance Test

The YBT protocol was adapted from Plisky et al., (2009) and Hertel et al., (2000). The YBT anterior, posterior-medial and posterior-lateral reaches were performed, in

that order, as one trial because CoP measures were recorded concurrently. Therefore, a composite score was equated from the greatest displacement of a single YBT trial, as oppose to a composite score of the greatest independent anterior, posterior-medial and posterior-lateral reach distances of separate YBT trials (as advocated by Plisky et al.,(2009) and Hertel et al., (2000)). Participants stood barefoot unilaterally on their non-dominant leg aligned with a centre line and a toe marker to ensure consistent start positions on the pressure mat. Participants were required to maintain the position of their stance foot throughout the test. Participants received six practice trials and three experimental trials to minimise any learning effect (Plisky et al., 2006, Hertel et al., 2000). Two minutes rest intervals were given between experimental trials. The YBT was standardised by placing hands on hips. Maintaining the unilateral stance position participants' reached with the non-standing limb in an anterior, posterior-lateral and posterior-medial sequence (in relation to the standing foot). The YBT profile was marked out with tape on the pressure mat and the floor. The posterior-lateral and posterior-medial lines were positioned at a 90^{o} angle to one another and at a 135^{o} angle from the anterior line (Plisky et al., 2009). All lines were marked out from the centre point to 160cm in length.

Participants received verbal instructions to: (1) reach with their non-standing leg 'as far as they possibly could under control', ensuring the movement and posture was maintained at all times; (2) touch the tape (marking out the YBT) lightly with the most distal part of their non-standing leg (toes of the foot); (3) ensure when they touched down with their toes they were not to bear any weight on the non-standing leg and that the pressure was to remain over the standing foot (base of support). A demonstration was provided by the investigator for each participant. The investigator

marked each point of contact during the YBT and then recorded each distance afterwards using a measuring tape. Participants YBT experimental trials were scored as a pass or a fail by the investigator. This was determined by correct execution of the YBT and whether all instructed criteria had been met. Each experimental trial that was scored as a pass was normalised to the participant's leg length (cm) (Equation 3.3). This was to allow for comparisons with subsequent testing as reach distance is related to limb length (Plisky et al., 2009). A YBT normalised composite score comprising the sum of the anterior, posterior-lateral and posterior-medial normalised reach distances was calculated and expressed as a percentage of leg length (Equation 3.4). Participant's best YBT normalised composite score was used for final analysis. CoP measures were recorded for each experimental trial.

Equation 3.3 (Gribble and Hertel, 2003)

*Reach Distance Normalised to Leg Length = (Reach Distance / Leg Length) * 100*

Equation 3.4 (Plisky et al., 2006)

*YBT Normalised Composite Score (% of Leg Length) = (Normalised ANT + PL + PM / 3 * Leg Length) * 100*

ANT = Normalised Anterior Distance; PL = Normalised Posterior-Lateral Distance; PM = Normalised Posterior-Medial Distance

3.3.6 Depth Jump Landing

Participants stood barefoot bilaterally either side of a centre line, shoulder width apart, with toes aligned with the leading edge of a 20cm plyometric box to ensure consistent start positions. DJL was standardised by placing hands on hips and the

drop from the plyometric box was always led with the non-dominant kicking leg. Participants received verbal instructions to: (1) 'step off the plyometric box leading with their non-dominant kicking leg'; (2) land two-footed on the force plate and stabilise as quickly as possible; (3) 'stick' their landing position until told by the investigator to relax. The investigator ensured each landing was held for three seconds. A demonstration was provided by the investigator for each participant. Participants received three practice trials and three experimental trials to minimise any learning effect. Two minutes rest intervals were given between the experimental trials. In the event of incorrect execution during all trials participants received corrective instructions via verbal feedback from the investigator. Any experimental trial that failed to meet the instructed criteria was negated. VGRF measures were recorded for each experimental trial. Participant's best TTS score from the three experimental trials was used for final analysis. TTS was determined by the time it took to dynamically stabilise VGRF (measured using a portable 2-axis force platform and SparkVue software) to within 5% of the participant's body weight (N) (Equation 3.5).

Equation 3.5: TTS 5% of Body Weight

$$5\%\ of\ Body\ Weight\ (N) = ((Body\ Mass * 9.80665) / 100) * 5$$

3.3.7 Foundation Performance Matrix Screen

The FPMS was employed by the football club to predict injury and performance and was conducted by the club's sport science staff who had received certified training to

do so. All FPMS tests were performed barefoot. (See Appendix 1.0 for the full FPMS and scoring criteria).

3.4 Statistical Analysis

All values presented in the results are reported as mean±*SD* and Cohen's *d* effect sizes (ES). Statistical analysis was performed using SPSS V.20 (IBM SPSS Statistics Version 20, IBM Company, UK). A one-way repeated measures ANOVA was used to test for significant group x time interactions and represented main effects. All post-hoc testing employed a bonferroni adjustment. In the event of a significant interaction a paired-samples t-test was run for each group. Independent samples t-test were run between groups for chronological age, APHV, maturational offset, body mass, stature, leg length, total game time and match frequency during the intervention and season to determine any significant difference (Table 3.1). Alpha was set at $P = 0.05$. Cohen's *d* ES was calculated from differences in means with the magnitude of effect recorded using Cohen's scale (Table 3.2) (Hopkins et al., 2009). Ninety-five percent confidence intervals (95% CI) were reported to represent the range of possible effect sizes.

Table 3.2: Cohen's scale for interpreting the magnitude of effect (Hopkins et al., 2009)

Effect Description	Effect Size
Trivial	0.0
Small	0.2
Moderate	0.6
Large	1.2
Very Large	2.0
Nearly Perfect	4.0
Perfect	Infinite

4.0 Results

4.1 Participant Characteristics

No significant difference was found between G1 and G2 for body mass (P = 0.128), stature (P = 0.734), leg length (P = 0.509), APHV (P = 0.056), intervention game time (P = 0.879), intervention match frequency (P = 0.442), season game time (P = 0.131), or season match frequency (P = 0.919) (Table 4.1). A significant difference was found between G1 and G2 for chronological age (P = 0.001) and maturational offset (P =0.001) (Table 3.1).

Table 4.1: Group mean±*SD* for total game time and matches played

	8-Week (In-Season) Intervention		Season 2013/14	
Group	**Total Game Time (mins)**	**Total Match Frequency**	**Total Game Time (mins)**	**Total Match Frequency**
G1	513.9 ± 225.4	7.3 ± 3.0	1797.9 ± 230.8	30.7 ± 6.0
G2	568.1 ± 136.5	6.8 ± 1.9	2190.9 ± 525.7	29.8 ± 7.2

4.2 Foundation Performance Matrix Measures

A significant group x time interaction was evidenced for FPMS Score (P = 0.036). No significant difference however was found pre and post FPMS Score for G1 (P = 0.235; ES = 0.180; 95% CI -1.94 - 0.60) or G2 (P = 0.80; ES =−0.136; 95% CI -0.12 -1.62) with only small ES changes in both (Figure 4.1). This indicates that the dose-response to G1 individualised DT and G2 generic DT had no difference on FPMS score.

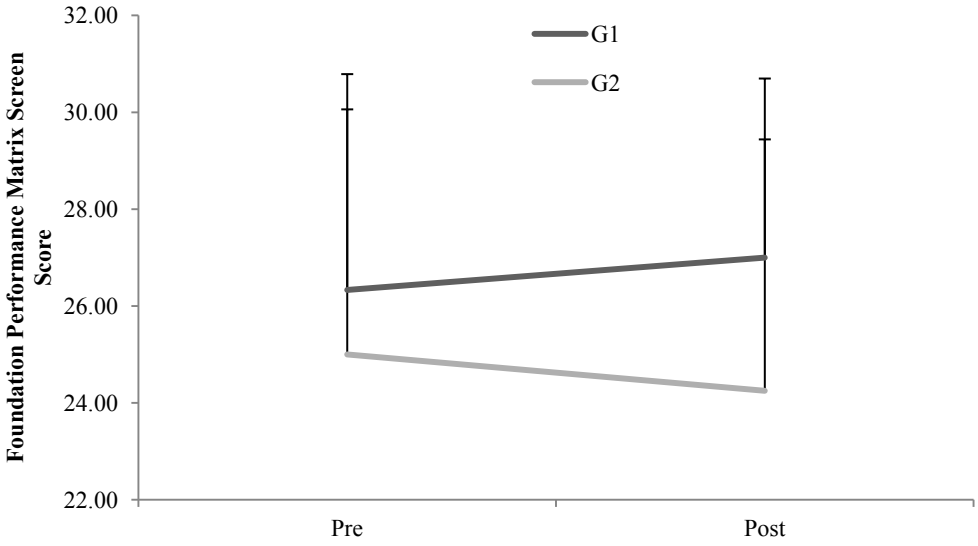


Figure 4.1: Group Mean±*SD* Foundation Performance Matrix Screen Scores Pre and Post Intervention

4.3: Depth Jump Landing

Peak VGRF (P = 0.474), loading rate (P = 0.335) and TTS (P = 0.637) (Figure 4.2) measures indicated no significant group x time interaction effects. Meaningful ES changes were observed for G1 and G2 (Table 4.2).

Table 4.2: DJL Measures

	G1					G2				
	Pre	**Post**	**ES**		**95% CI**	**Pre**	**Post**	**ES**		**95% CI**
Peak VGRF (N)	2006.14 ± 346.6	1797.57 ± 232.84	Moderate	0.706	1725.35 / 2078.36	1808.28 ± 281.28	1782.51 ± 256.29	Trivial	-0.096	1642.54 / 1948.25
Loading Rate (ms)	73.17 ± 9.83	51.17 ± 13.19	Large	-1.89	0.046 / 0.079	79.13 ± 25.28	81.50 ± 39.40	Trivial	0.072	0.067 / 0.095
TTS (ms)	727.83 ± 423.94	446.5 ± 99.56	Moderate	-0.914	0.391 / 0.786	723.00 ± 203.97	529.25 ± 217.68	Moderate	-0.919	0.455 / 0.797

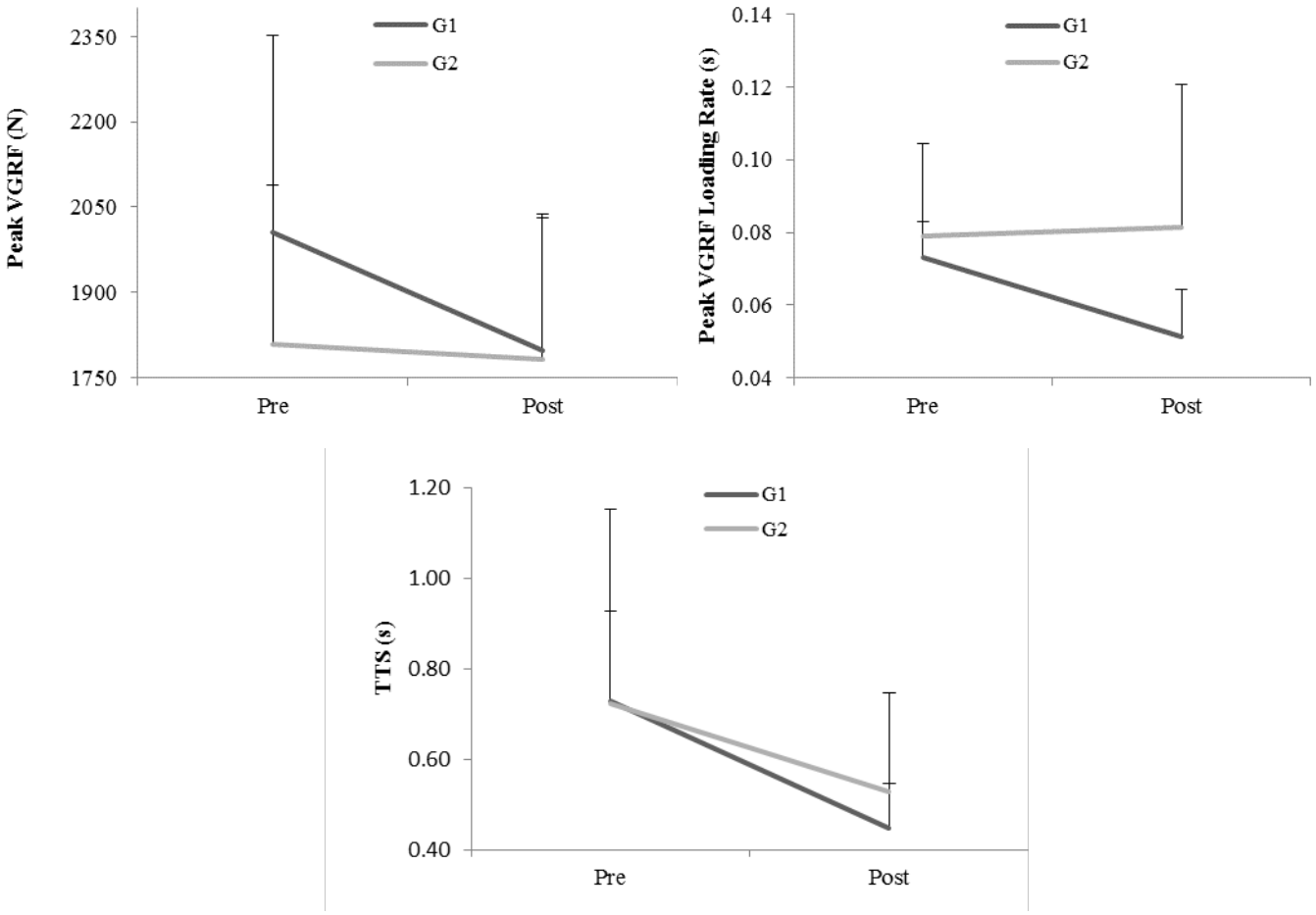


Figure 4.2: Group Mean±*SD* Peak VGRF, Peak VGRF Loading Rates and TTS from DJL trials used for final analysis

4.4: Y Balance Test

No significant group x time interactions were found for G1 or G2 YBT CoP Total Displacement (P = 0.295), Sway Velocity (P = 0.235), CoP X Displacement (P = 0.205), CoP Y Displacement (P = 0.112), or Ellipse Area (P = 0.981) (Table 4.3).

Table 4.3: Group Mean±*SD* YBT CoP Measures

	G1					G2				
	Pre	**Post**	**ES**		**95% CI**	**Pre**	**Post**	**ES**		**95% CI**
Total Displacement (cm)	124.14 ± 13.26	132.05 ± 19.87	Small	0.488	113.32/ 142.87	120.54 ± 16.43	118.33 ± 18.69	Trivial	-0.126	106.65 / 132.23
Sway Velocity (cm·s-^{1})	8.76 ± 0.91	9.84 ± 1.53	Moderate	0.858	8.30 / 10.30	9.19 ± 1.50	9.14 ± 1.11	Trivial	-0.034	8.30 / 10.03
***X* Displacement (cm)**	16.71 ± 3.10	17.71 ± 2.16	Small	0.375	14.36 / 20.09	20.11 ± 6.31	16.74 ± 2.91	Moderate	-0.687	15.93 / 20.92
***Y* Displacement (cm)**	5.96 ± 1.70	5.08 ± 0.63	Moderate	-0.681	4.69 / 6.35	5.57 ± 0.77	6.36 ± 1.40	Moderate	0.693	5.24 / 6.68
Elipse Area (cm^{2})	45.33 ± 57.92	74.01 ± 133.85	Small	0.278	-43.52 / 162.86	111.09 ± 111.96	141.21 ± 145.79	Small	0.232	36.79 / 215.52

The YBT normalised composite score (P = 0.181) (Figure 4.3) indicated no significant group x time interaction effects, with only moderate and small ES observed for G1 and G2 respectively (Table 4.4). Despite YBT independent anterior (P = 0.908), posterior-lateral (P = 0.430) and posterior-medial (P = 0.106) reach directions indicating no significant group x time interaction effects, meaningful ES were observed for G1 and G2 (Table 4.4).

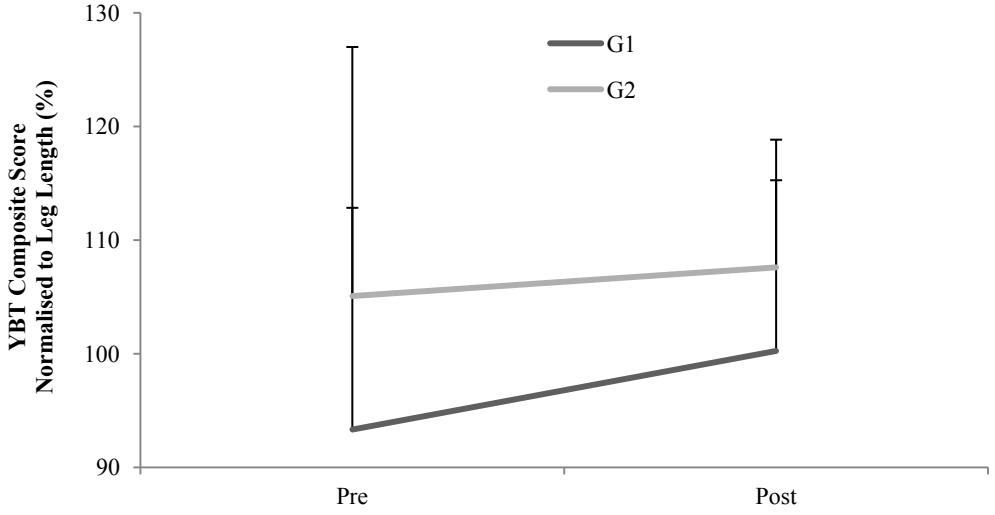


Figure 4.3: Group Mean±*SD* YBT Normalised Composite Score

Table 4.4: Group Mean±*SD* YBT Normalised Composite and Independent Reach Distances

	G1					G2				
	Pre	**Post**	**ES**		**95% CI**	**Pre**	**Post**	**ES**		**95% CI**
Normalised Composite Score	93.32 ± 7.39	100.24 ± 6.47	Moderate	0.996	90.78 / 102.78	105.08 ± 7.65	107.59 ± 5.52	Small	0.376	101.14 / 111.53
Normalised Anterior Reach	67.22 ± 4.05	72.37 ± 3.17	Large	1.416	66.30 / 73.29	67.12 ± 5.20	72.62 ± 4.75	Moderate	1.104	66.84 / 72.90
Normalised Posterior-Lateral Reach	91.10 ± 8.56	99.09 ± 7.50	Moderate	0.993	86.90 / 103.30	105.65 ± 12.47	110.21 ± 6.91	Small	0.452	100.83 / 115.03
Normalised Posterior-Medial Reach	94.03 ± 9.78	100.33 ± 4.85	Moderate	0.816	91.48 / 102.88	106.69 ± 7.42	104.81 ± 5.92	Small	-0.280	100.81 / 110.69

4.5: Single Leg Stance Test

No significant group x time interactions were found for SLST CoP Total Displacement (P = 0.223), Sway Velocity (P = 0.132), CoP X Displacement (P = 0.435), CoP Y Displacement (P = 0.286), or Ellipse Area (P = 0.454) (Table 4.5). No meaningful ES changes were observed for G1 or G2.

Table 4.5: SLST CoP Measures

	G1					G2				
	Pre	**Post**	**ES**		**95% CI**	**Pre**	**Post**	**ES**		**95% CI**
Total Displacement (cm)	201.55 ± 36.11	185.76 ± 39.46	Small	-0.417	165.65 / 221.66	162.86 ± 31.19	171.07 ± 27.46	Small	0.280	142.71 / 191.22
Sway Velocity (cm·s-^{1})	3.43 ± 0.61	3.16 ± 0.67	Small	-0.417	2.83 / 3.76	2.77 ± 0.54	2.98 ± 0.39	Small	0.450	2.47 / 3.28
***X* Displacement (cm)**	5.76 ± 1.47	5.85 ± 1.52	Trivial	0.061	2.69 / 8.93	5.62 ± 1.33	9.06 ± 8.71	Small	0.552	4.64 / 10.04
***Y* Displacement (cm)**	4.31 ± 0.98	4.08 ± 0.63	Small	-0.277	2.38 / 6.00	3.71 ± 0.45	5.90 ± 4.83	Moderate	0.640	3.24 / 6.38
Elipse Area (cm^{2})	2.88 ± 1.33	2.25 ± 1.07	Small	-0.518	1.66 / 3.47	2.46 ± 1.08	2.31 ± 0.87	Trivial	-0.152	1.60 / 3.17

5.0 Discussion

5.1 Main Findings

Irrespective of no significant changes, eight weeks DT had meaningful effects on neuromuscular control. The magnitudes of effects however were greater for G1 than G2. As SLST, YBT and DJL indicated greater effects than the FPMS and have all been proposed to predict injury, they could be a suitable surrogate marker for assessing the effects of DT. These findings also suggest that a lower dose of DT is sufficient for improving neuromuscular control provided training is individualised.

5.2 The Effects of DT on Kinematic and Kinetic Assessments of Motor Control

i. Foundation Performance Matrix Screen

The findings of this research are novel as there is no empirical literature supporting the efficacy of the FPMS in an athletic environment and only limited research supporting DT. The small positive and negative effects on FPMS score for G1 and G2 respectively indicates that one individualised DT session per week is sufficient for improving FPMS performance. The observed eight week dose-response for G1 and G2 also indicates that the stimulus presented to elicit a neural adaptation is lacking when prescribed generically, irrespective of a greater training frequency. This further advocates that the efficacy of remedial motor control DT strategies is specific to an individual's needs. Comparably Danneels et al., (2001) found that 10

weeks generic DT targeting the multifidus muscle, performed three times weekly, failed to increase cross-sectional area in the absence of an additional higher-threshold stimulus. Whereas training comprising DT as well as dynamic- and static-strength resistance training significantly increased multifidus cross-sectional area. While DT focally presents a neural stimulus to remedy specific motor control dysfunctions, a degree of muscular hypertrophy might be expected following the reciprocal-activation of select musculature during cognitive dissociated movement (Mottram and Comerford, 2008, Mottram et al., 2005). Danneels et al., (2001) adhered to Mottram and Comerford (2001a) and Comerford and Mottram (2008) DT guidelines, prescribing 15 to 18 repetitions of recruitment specific exercises with five second isometrics at active end-range of motion. When considering the exercises specifically targeted the multifidus, the multifidus's accumulative time-under-tension is comparable with other studies where hypertrophic adaptations have been observed (Schoenfeld et al., 2015, 2004, Campos et al., 2002). However, this can only be inferred as this study did not measure cross-sectional area. In addition, Mottram and Comerford (2001a) postulated the tonic activation of local stabilising musculature required to remedy dynamic postural control was circa 25% of maximum. Danneels et al., (2001) had specified that the tonic multifidus activation during the prescribed DT exercises was circa 30% of maximum activation. In support of this study, Danneels et al., (2001) findings reinforce that although identified sites of dysfunction may be the common amongst homogenous individuals, as evidenced by the FPMS for G2, the causation of motor control dysfunction is independent to the individual. This would be in agreement with Mottram and Comerford (2001a) notion that the efficacy of correcting motor control is dependent on challenging cognitive processes and afferent feedback. Individualised DT would therefore better facilitate this

elucidating G1 superior enhancement in motor control compared to G2. Danneels et al., (2001) did not use the FPMS to direct DT. This therefore supports Mottram and Comerford (2008) premise that the FPMS can identify both the site and direction of the dysfunction. Similarly, Wright et al., (2015) found trivial effects (13% beneficial/ 82% trivial/ 5% harmful, 0.2 ± 1.2 90% CL) based on adjusted change scores and no smallest worthwhile effect (= >0.2 ±*SD*) in adolescents matched by FMS composite score following four weeks of either four 30 minute fundamental movement skills or generic multi-sport training per week. Like the FPMS and its associated DT, the fundamental movement skills training was directed according to FMS composite score and its associated independent advanced corrective exercise manual (Cook, 2010). However, comparable to G2 evidence, Wright et al., (2015) findings infer that the dose-response to generic short-term independent motor control training had negligible effects on reducing injury or attenuating compensatory synergies. This is important because sport-specific kinematics can elicit synergistic dominant motor patterns and hypertrophic cross-sectional area adaptations (Hides et al., 2012, Jakobsen et al., 2011, Hides et al., 2008, Ranson et al., 2008, Engstrom et al., 2007, Hides et al., 2007, Lucki and Nicolay, 2007), as evidenced by Kearns et al., (2001) in English male elite academy footballers lower limb musculature. However, G1 results advocate that individualised DT prescribed according to an individual's FPMS performance profile can attenuate potential footballing induced synergistic motor skills.

This study and the aforementioned evidence advocate that the efficacy of motor control retraining strategies is dependent on individualisation. However, Tsao and Hodges (2007) isolated-DT and sit-up protocols, designed to immediately effect

postural control through enhanced anticipatory postural adjustments, decreased Transverses Abdominis onset of activation timing, and increased activation amplitude. Despite focussing on low-threshold tonic activation of specific muscles enhancing local stabilisation, the DT evidenced no significant difference with the higher-threshold sit-up protocol (which would have involved the high-threshold phasic recruitment of local as well as global stabilising and mobilising musculature), comparable to Wright et al., (2015). In addition, there was no change in the remaining six SEMG sites, or any indication of anticipatory postural adjustments adaptations for either protocol. Although no change in anticipatory postural adjustments was found, the Transverses Abdominis earlier onset of activation and greater amplitude indicates a desirable adaptation. This is because the Transverses Abdominis is considered central to regulating the transfer of force through the kinetic chain (Kibler et al., 2006, Hodges and Moseley, 2003, Hodges and Richardson, 1997, Hodges and Richardson, 1996). However, Tsao and Hodges (2007) investigated a population with no training history. As non-athletes who were injury free they would have not likely developed any chronic dominant or compensatory synergistic motor patterns from sport-specific kinematics (Ngomo et al., 2015, Hides et al., 2012, Jakobsen et al., 2011, Hides et al., 2008, Ranson et al., 2008, Engstrom et al., 2007, Hides et al., 2007, Lucki and Nicolay, 2007, Hodges and Moseley, 2003, Sterling et al., 2001, O'sullivan, 2000). The scope for any acute or chronic alteration in motor output therefore would have been much greater than that of the trained academy footballers in G1 and G2 of this study (Behm et al., 2002). Likewise, because of the Transverses Abdominis dynamic function changes in activation onset and amplitude are expected during both tonic and phasic recruitment (Kibler et al., 2006, Sahrmann, 2002, Comerford and Mottram, 2001a,

Umphred et al., 2001, Janda, 1993). For instance, although DT retrains the local stability system through tonic, low-threshold isolated muscle activation (Mottram and Comerford, 2008, Comerford and Mottram, 2001a), the synergistic co-activation of global stabilising musculature through phasic, high-threshold activation is inevitable during dynamic movement (Kibler et al., 2006, Sahrmann, 2002, Comerford and Mottram, 2001a, Umphred et al., 2001, Janda, 1993). However, as Tsao and Hodges (2007) found no changes other than for the Transverses Abdominis this reinforces the findings of this study that only individualised DT can elicit chronic motor output adaptations. G1 evidence also infers that while DT can enhance motor control, the efficacy of individualised DT dose-response is dependent on FPMS direction. Caution should be exercised though when using the FPMS to direct DT to lower the risk of injury in elite academy footballers because the FPMS comprised three upper body assessments (Table 2.5). As 90% of reported non-contact injuries are to the lower extremity in elite academy footballers (Ekstrand et al., 2011, Hagglund et al., 2009, Price et al., 2004) (Table 2.2) the suitability of using the FPMS as an injury screen must be questioned.

ii. Depth Jump Landing

G1 evidenced superior whole-body stabilisation and a greater ability to attenuate PVGRF during DJL, which indicates greater transfer of GRFs through the kinetic chain, symptomatic of enhanced LPHC stabilisation (Kibler et al., 2006, Hodges and Moseley, 2003, Hodges and Richardson, 1997, Hodges and Richardson, 1996). Although G1 and G2 both decreased DJL PVGRF, G1 indicated a greater magnitude

of change. Moreover, while G1 and G2 TTS performance improved by the same magnitude, a large effect was found for the reduction in G1 PVGRF loading rate whereas G2 increased. As the demand placed on the musculoskeletal structures to attenuate force increases synchronously with PVGRF loading rates (Flanagan et al., 2008), increased TTS and PVGRF loading rates have been evidenced to increase the risk of non-contact injury (Fong et al., 2011, Kulig et al., 2011, Zadpoor and Nikooyan, 2011, Yeow et al., 2009, Oliver et al., 2008, Yu et al., 2006, Hewett et al., 2005, Hreljac et al., 2000). The large disparity between G1 and G2 DJL performance therefore reinforces the dose-response impact of individualised DT. Furthermore, when compared with empirical literature investigating DJL, this study's evidence infers that individualised DT does reduce the risk of injury. For instance, Hewett et al., (2006) found that circa- and post-pubertal male athletes with greater LPHC motor control evidenced decreased VGRF and knee valgus torque during 31cm bilateral DJL. In an earlier study Hewett et al., (2005) had also evidenced that greater knee abduction moments correlated with increased PVGRF (r = 0.88, P = 0.001). Hewett et al., (2006) and Hewett et al., (2005) findings therefore advocate that the DT intervention in this study enhanced LPHC stabilisation. Furthermore, although G1 and G2 reduced TTS and PVGRF, the DJL assessment identified that only G1 decreased PVGRF loading rate.

G1 and G2 evidenced capability to attenuate PVGRF following DT would also reduce the work-load placed on the MTU, delaying the onset of neuromuscular fatigue. This would therefore reduce the potential manifestation of overuse injuries (Mersmann et al., 2014, Couppé et al., 2013, Hansen et al., 2013, Arya and Kulig, 2010, Stafilidis and Arampatzis, 2007, Arampatzis et al., 2006). This is highlighted

by Oliver et al., (2008) investigation into 20cm bilateral DJL performance following a 42 minute intermittent fatiguing protocol simulating one half of match play. Using the same drop height and players of comparable chronological age (15.8 ± 0.4 years) and physical characteristics to G1 (body mass 59.8 ± 9.7 kg, stature 1.73 ± 0.06m) Oliver et al., (2008) found that PVGRF increased (pre 1862 ± 429N vs. post 1889 ± 429N) and impact GRF significantly ($P < 0.05$) (pre 2135 ± 369N vs. post 2499 ± 422N) post simulated match play. Oliver et al., (2008), Hewett et al., (2006) and Hewett et al., (2005) evidence, as well as G1 and G2 findings all advocate that the DJL could be a valuable surrogate monitoring measure of DT. In addition, the DJL offers immediate kinematic and objective kinetic feedback (Wikstrom et al., 2004) and has been previously evidenced as an appropriate measure in youth populations (Oliver et al., 2008, Hewett et al., 2006, Barber-Westin et al., 2005). Furthermore, using the DJL as a surrogate DT monitoring measure has practical applications for elite academy footballers. This is because an individual's normative DJL data could be used to measure their pre training and/or competition neuromuscular fatigue status owing it the relationship with motor output (Cormie et al., 2011, Allen et al., 2008, Millet and Lepers, 2004, Gandevia, 2001). It does remain to be elucidated whether the absence of the FPMS performance profile to direct individualised DT prescription would affect the efficacy of DT to reduce injury risk. Nonetheless, G1 evidence does indicate that the DJL assessments immediate kinematic and kinetic feedback could be an effective high-threshold surrogate monitoring measure of DT.

iii. Y Balance Test

Increased excursion distances on the YBT represent superior motor control, strength, stabilisation and active range of motion at the ankle, knee and LPHC (Munro and Herrington, 2010, Hale et al., 2007, Hertel et al., 2006, Plisky et al., 2006, Gribble et al., 2004, Olmsted et al., 2002, Earl and Hertel, 2001). Like the DJL performance, G1 indicated consistently greater positive magnitudes of change for all YBT measures, with G2 indicating a decrease in performance for posterior-medial reach distance. Potential adaptations in motor control are further reflected by the YBT CoP measures. For instance, while the greater total and *X* displacements for G1 are expected because of their enhanced YBT performance, the concomitant increase in sway velocity infers that G1 enhanced motor control is symptomatic of compensatory postural adjustments rather than anticipatory postural adjustments adaptations (dos Santos et al., 2014, Takacs et al., 2014, Knapp et al., 2011, Ruhe et al., 2010, Ross et al., 2009, Voight et al., 1998). This is indicated by the magnitude of the COM's displacement and velocity feedback (Masani et al., 2003). As greater YBT excursion distances indicate enhanced LPHC stabilisation, and therefore motor control (Filipa et al., 2010, Munro and Herrington, 2010, Plisky et al., 2009, Hale et al., 2007, Plisky et al., 2006, Gribble et al., 2004, Olmsted et al., 2002, Earl and Hertel, 2001), the concomitant increase in sway velocity is likely associated with the electromechanical delay in activation synonymous with compensatory postural adjustments pathways (dos Santos et al., 2014, Masani et al., 2003, Voight et al., 1998). Conversely, while sway velocity decreased for G2 during the YBT this is likely attributable to the negative effects for total and *X* displacements respectively,

symptomatic of reduced motor control. However, this can only be inferred as electromechanical delay was not recorded and therefore remains to be elucidated.

Impaired afferent function at the ankle has previously been evidenced to limit CoP sway displacement and therefore sway velocity (dos Santos et al., 2014, Basnett et al., 2013, Kiers et al., 2012). In particular, reciprocal afferent pathways between the ankle and the LPHC have been evidenced to aid COM stabilisation (dos Santos et al., 2014, Kiers et al., 2012), which is central to enhanced motor control (Filipa et al., 2010, Mottram and Comerford, 2008, Zazulak et al., 2007, Hewett et al., 2006, Won and Hogan, 1995). Although G2 YBT normalised composite score improved post intervention, the greater magnitude of change for G1 infers that the dose-response to individualised DT had a greater effect on afferent sensory function. This notion is supported by the compensatory postural adjustments velocity feedback and enhanced LPHC stabilisation observed for G1. The reciprocal afferent synergies of the ankle and LPHC may also elucidate the decrease in G2 posterior-medial reach distance. For instance, Hubbard et al., (2007a) found that chronic ankle instability significantly correlated with decreased posterior-medial (r = .84, P = 0.001) and anterior (r = .65, P = 0.001) reach distances, accounting for 71% and 42% of the variance for decreased reach distance. This suggests that chronic ankle instability and/or impaired afferent function could have been a contributing factor as to why G2 YBT normalised composite performance only marginally improved (Table 4.4). This can however only be surmised. Likewise, Hubbard et al., (2007b) found increased hip abduction significantly (P < 0.05) correlated with increased hip extension strength (r = .70), accounting for 26% of the variance, and increased posterior-medial excursion distance (r = .51), accounting for 49% of the variance. As balanced

synergist-antagonist intermuscular coordination stabilises joints against perturbation, force generation (i.e., the expression of strength) is also enhanced as the electromechanical delay of resisting forces decreases eliciting earlier onset and activation pattern (Anderson and Behm, 2005, Behm et al., 2002, Lloyd, 2001, Kearney and Hunter, 1990). DT has been purported to enhance motor activation onset and pattern of recruitment (Mottram and Comerford, 2008, Comerford, 2006, Comerford and Mottram, 2001a, Comerford and Mottram, 2001b, Danneels et al., 2001). Both are central to maintaining postural control during dynamic movements and force generation (Anderson and Behm, 2005, Behm et al., 2002). The potential causes for G2 reduced YBT performance however can only be surmised from comparable investigations. Nonetheless, this study does indicate that the dose-response to, and the efficacy of the generic DT intervention to enhance motor control and reduce the risk of injury was inferior compared to G1 individualised DT.

This finding is supported by Plisky et al., (2006). Using 130 male high-school basketball players of comparable age (14 to 18 years) to the academy footballers in this study, Plisky et al., (2006) used the YBT to predict lower extremity injury. When normalised to leg length Plisky et al., (2006) found that individuals with a YBT composite score of less than 94% of leg length increased the risk of lower extremity injury three-fold. Figure 4.3 shows that G1 pre DT intervention YBT normalised composite score of 93.32 ± 7.39% means they were three-times more likely than G2 (who had a pre intervention YBT normalised composite score of 105.08 ± 7.65%) of sustaining a lower extremity injury. This was compounded by the fact that players comprising G1 and G2 were already at high risk of sustaining a lower extremity injury as they account for over 90% of reported non-contact injuries

in elite academy football (Ekstrand et al., 2011, Hagglund et al., 2009, Price et al., 2004). However, the observed magnitude of positive effects for G1 was consistently greater than G2 for YBT normalised composite score and independent reach distances. Interestingly, based on Plisky et al., (2006) evidence G1 dose-response to individualised DT reduced their risk of lower extremity injury by three-fold. However, when compared with Filipa et al., (2010) investigation into adolescent female footballers (aged EXP: 15.4 ± 1.5 years) YBT performance post an eight week multifaceted Integrative Neuromuscular Control Training intervention performed twice-weekly, their dose-response was superior to G1 individualised and G2 generic DT. This was despite both studies motor control training focussing on enhancing trunk stabilisation and lower extremity strength expression over eight weeks. Unlike G1 and G2 DT, Filipa et al., (2010) Integrative Neuromuscular Control Training comprised high- and low-threshold stable and unstable proprioceptive, plyometric, and strength and power resistance training exercises. In addition, unlike G1 and G2, post YBT normalised composite score increased significantly on the left (pre 96.9% ± 10.1% & post 103.4% ± 8.0%; $P = 0.04$) and right (pre 96.4% ± 11.7% & post 104.6% ± 6.1%; $P = 0.03$) legs. Posterior-lateral reach distance on the right ($P = 0.008$, η^2 ES 0.41) and left ($P = 0.04$, η^2 ES 0.27) leg, as well as posterior-medial reach distance on the left leg ($P = 0.03$, η^2 ES 0.3) also increased significantly and evidenced large ES (partial η^2). By comparison, Table 4.4 shows that G1 and G2 ES for YBT normalised composite score and posterior-lateral excursions were only moderate (ES = 0.996 & ES = 0.993) and small (ES = 0.376 & ES = 0.452) respectively. While G1 ES for posterior-medial excursion was still only moderate (ES = 0.816), G2 had a small negative effect (ES = -0.280). This indicates that multifaceted programmes comprising exercises can

enhance motor control and LPHC stabilisation. Furthermore, Filipa et al., (2010) findings reinforce that G2 generic DT was ineffective. Especially owing to the smaller magnitudes of change observed for G2 YBT performance despite having a greater training frequency over the same intervention period of eight weeks. However, DT only comprised specific low-threshold exercises focussed on tonic activation. When considering the dose-response to G1 individualised DT resulted in moderate to large effects on YBT performance from only one DT session per week, this suggests that individualised DT could be even more effective when prescribed within a multifaceted programme. Caution should be exercised however when directly comparing the efficacy of respective training modalities as Filipa et al., (2010) investigated female amateur footballers. Females have been reported to demonstrate different recruitment synergies to males (Hewett et al., 2006). In addition, the scope for any adaptation in motor control would have arguably been greater in amateurs than elite academy footballers (Behm et al., 2002).

iv. Single Leg Stance Test

All the CoP measures indicated that G1 SLST performance improved whereas G2 SLST performance decreased post their respective eight weeks DT. The only exceptions to this were G1 greater *X* displacement (cm) and G2 decreased ellipse area (cm^2), although both only indicated trivial effects (Table 4.5). In addition, although G2 only indicated small and moderate effects for *X* and *Y* displacement's their post testing standard deviation increased circa eight- and ten-fold respectively. This infers a heterogeneous dose-response to the generic DT. When considering

Matsuda et al., (2008) inverted pendulum theory during unilateral stance, the small magnitude of change for G2 increased sway velocity is further symptomatic of a reduced response to generic DT. This is because the greater mass of the torso over the narrowed BOS would result in faster accelerations of the COM in the absence of enhanced anticipatory postural adjustments and compensatory postural adjustments motor control (dos Santos et al., 2014, Barone et al., 2011, Matsuda et al., 2008). While this study did not investigate electromechanical delay (during select muscle synergies to determine anticipatory feed-forward and compensatory feedback postural adjustment pathways), Matsuda et al., (2008) theory advocates the small effect for G1 decreased sway velocity is symptomatic of enhanced motor control, as oppose to G2. Furthermore, plantar CoP measures are proportional to ankle torque generated by the body's COM displacement. Thus, plantar CoP measures purportedly reflect the effectiveness of the ankle and LPHC reciprocal synergies to regulate static balance through joint stiffness during the SLST (Abdul Razak et al., 2012, Balasubramaniam and Wing, 2002, Baratto et al., 2002, Gatev et al., 1999, Winter, 1995). . This is significant as compared to their amateur counterparts and other athletes, elite academy and senior footballers have evidenced superior motor control during unilateral stance (Pau et al., 2014, Barone et al., 2011, Matsuda et al., 2008, Paillard et al., 2006, 2004). Furthermore, elite academy and senior players have evidenced lower *X* and *Y* sway displacement on the non-dominant/ stance leg compared to the dominant kicking leg (Pau et al., 2014, Barone et al., 2011, Matsuda et al., 2008, Paillard et al., 2006, 2004). This is purportedly because of the increased afferent workload due to the greater postural control required to execute the complex motor skill of kicking a football from a unilateral stance (Kellis and Katis, 2007, Kellis et al., 2001). The scope for any adaptation in motor control therefore would

invariably be expected to be less in elite academy footballers. As this study only investigated the non-dominant/ stance leg this further illustrates the magnitude of G1 dose-response to one session of individualised DT per week compared to G2 three sessions of generic DT. Table 4.5 also indicates that G1 and G2 sway velocity during the SLST was greater than both the adult regional (2.17 ± 0.46 cm·s-^{1}) and national (1.67 ± 0.47 cm·s-^{1}) footballers investigated by Paillard et al., (2006). As national players trained every day vs. regional players training twice a week, national players would be expected to have a greater dose-response and therefore enhanced motor control, evidenced by the lower sway velocity. The opposite however was found for this study. Despite G2 training three-times per week compared to G1 one (technical football training and DT), G1 evidenced superior motor control adaptations over eight weeks. However, the origins of the national players enhanced motor control remains to be elucidated as Paillard et al., (2006) only specified training frequency rather than what a training day comprised. Therefore, it is unknown whether motor control enhanced through exposure to football training/ competition and/ or physical competency based training modalities. There are inherent differences between adolescents and adults neurological pathways, as well as between circa- and post-pubescents (Lazaridis et al., 2010, Oliver and Smith, 2010, Lambertz et al., 2003). These factors therefore require consideration when comparing the results of this study.

The findings of Pau et al., (2014) investigation into male elite academy footballers (age 14.5 ± 0.2 years, stature 164.5 ± 5.6 cm, body mass 56.8 ± 6.8 kg) has the greatest implications when compared with this study. This is because to the authors knowledge Pau et al., (2014) investigation is the only study to have conducted a

SLST congruently with plantar CoP measures in elite male academy footballers. Pau et al., (2014) used an RS Footscan pressure mat to record CoP measures from a SLST pre and post Buchheit et al., (2010a) specific adolescent footballers RSA protocol simulating match fatigue. Pau et al., (2014) found CoP ellipse area (cm^2) maximum total (63.7 ± 15.9 vs. 72.1 ± 18.2 cm), anterior-posterior (3.5 ± 1.4 vs. 4.2 ± 1.9 cm) and medial-lateral (2.2 ± 7.2 vs. 2.9 ± 0.9 cm) displacements, and sway velocities ($cm \cdot s^{-1}$) all significantly ($P < 0.05$) increased (medial-lateral sway velocity ($cm \cdot s^{-1}$) also increased compared to baseline values but not significantly). However unlike Barone et al., (2011), Matsuda et al., (2008) and Paillard et al., (2006), Pau et al., (2014) found no interaction between the dominant and non-dominant kicking leg pre or post. This could possibly be a reflection of player's technical ability as invariably they would be expected to execute football-specific motor skills with both feet, as well as been exposed to supervised physical training at elite academy level (Barone et al., 2011, Matsuda et al., 2008, Paillard et al., 2006). Positive correlations between the fatigue-index and sway-fatigue/rest-ratio for CoP total displacement (cm) ($r = 0.631$, $P = 0.01$), anterior-posterior sway velocity ($cm \cdot s^{-1}$) ($r = 0.577$, $P = 0.01$) and medial-lateral sway velocity ($cm \cdot s^{-1}$) ($r = 0.529$, $P = 0.014$) however were evidenced for the non-dominant/ stance leg. This indicates that the non-dominant leg's rate of fatigue during match play was greater than the dominant kicking leg, and that sway displacement and velocity increase exponentially with neuromuscular fatigue. Pau et al., (2014) findings therefore advocate that based on this study's post intervention data G2 are at greater risk of injury than G1. This is because unlike G1 individualised DT, the generic DT prescribed to G2 had a detrimental impact on motor control. Pau et al., (2014) findings therefore reinforce that G2 would be at greater risk of injury than G1, and

that G2 generic DT was ineffective compared to G1 individualised DT as it had a detrimental impact on SLST performance and plantar CoP measures. Increased CoP sway displacements and velocities during static unilateral stance is symptomatic of decreased motor control (Takacs et al., 2014, Abdul Razak et al., 2012, Knapp et al., 2011, Ross et al., 2009, Santos and Aruin, 2009, Hubbard et al., 2007b, Mochizuki et al., 2006). In addition, neuromuscular fatigue is a pathological factor of acute and overuse non-contact injuries accounting for 90% of injuries in English elite academy football (Ekstrand et al., 2011, Hagglund et al., 2009, Price et al., 2004). Alarmingly, the range of G1 and G2 *X* and *Y* displacement is also closer to Pau et al., (2014) post RSA protocol fatigued values. The elite academy players in Pau et al., (2014) study trained for a minimum of six hours per week. By comparison, G1 symptomatic decreased motor control may be expected due to a lower weekly training frequency (despite having a greater dose-response effect than G2). However, G2 training was greater than six hours a week suggesting inappropriate training prescription. However, like Paillard et al., (2006), Pau et al., (2014) did not state what a training day comprised.

5.3 The Effects of Maturation and Growth on the Mechanisms Underpinning the Dose-Response to DT and Assessments of Motor Control

As would be expected, G1 and G2 maturational offset from predicted APHV was significantly different ($P = 0.001$) and indicated that G1 were circa puberty whereas G2 were post-pubescent (Table 3.1). Inherent musculoskeletal and neurophysiological differences between them therefore could elucidate G1 and G2

dose-response to DT, as well as their respective FPMS, DJL, YBT and SLST performances (Mersmann et al., 2014, Meylan et al., 2014, Couppé et al., 2013, Hansen et al., 2013, Arya and Kulig, 2010, Lazaridis et al., 2010, Meylan et al., 2010, Oliver and Smith, 2010, Xu et al., 2009, Beunen and Malina, 2008, Grosset et al., 2007, Croce et al., 2004, Malina et al., 2004, De Ste Croix et al., 2003, Lambertz et al., 2003, Hawkins and Metheny, 2001, Gasser et al., 2001, Malina et al., 2000, Viru et al., 1999). When comparing predicted APHV, Lloyd et al., (2015) and Le Gall et al., (2007) investigations using academy players from UK professional clubs (Table 2.3) and the French Centre of Excellence (13.3 ± 0.3 years, range 12.3 to 14.4) indicate that G1 and G2 were late maturers. Le Gall et al., (2007) also evidenced that late maturers had a lower incidence of moderate injuries compared to normal and early maturers. Therefore, G1 and G2 inherent slower growth and maturational rate could abate the intensification of injury risk, notably during circa pubescent periods (Beunen and Malina, 2008, Gasser et al., 2001, Hägg and Taranger, 1991). Johnson et al., (2009) general log linear analysis also concluded maturation, as well as competition and training exposure, accounted for 47% of the variance in injury incidence. Identifying APHV to establish early, normal, or late maturational status is therefore not only important for determining the level of risk (Le Gall et al., 2007), but also when using physical competency assessments of motor control to predict injury risk. For instance, Lloyd et al., (2015) found that despite U16 advanced maturational status, and significantly ($P < 0.05$) outperforming U13 and U11 on the FMS (Table 2.3), ANCOVA applied to maturation indicated no significant between group differences. This indicates that the screen outcome was influenced by maturational status. Moreover, the FMS inline lunge, active straight leg raise, and rotary stability assessments are biomechanically

comparable to the FPMS tests three, five, seven and nine (Table 2.5). This is conjectural however and the effect of different stages of biological development on the FPMS remains to be elucidated. Nonetheless, as changes in FPMS outcome are considered reflective of DT efficacy (Mottram and Comerford, 2008), any change in motor control that is, to a larger or lesser extent, a result of growth and/or maturation means that the effectiveness of the prescribed DT could be misinterpreted.

Concomitant increases in limb mass and moment of inertia with long bone growth, in the absence of MTU hypertrophy and ligament cross-sectional area, lead to earlier onset of neuromuscular fatigue (Mersmann et al., 2014, Xu et al., 2009, Stafilidis and Arampatzis, 2007, Arampatzis et al., 2006, Hawkins and Metheny, 2001). Invariably this reduces joint stiffness and increases the risk of chronic or acute overload injuries (Mersmann et al., 2014, Xu et al., 2009, Stafilidis and Arampatzis, 2007, Arampatzis et al., 2006, Hawkins and Metheny, 2001). Furthermore, the greater force requirement would increase joint torque when the connective tissue tensile strength is already close to the failure limits as a consequence of the increased pre-load (Mersmann et al., 2014, Couppé et al., 2013, Hansen et al., 2013, Arya and Kulig, 2010, De Ste Croix et al., 2003). This evidence advocates that the risk of injury will exponentially reflect the rate that growth and maturational changes occur. G1 anthropometric measures and post-APHV circa-pubescent status (Table 3.1) infer that they might have been at greater risk of injury through these mechanisms. No significant difference between G1 and G2 stature and leg length (which accelerates before APHV (Beunen and Malina, 1988)) also advocates that G1 growth spurt had already occurred. As late maturers however, G1 inherent slower bone growth and maturational rate would offset the degree of risk because the pre-load on the MTU

would be less than that for normal and early maturers (Xu et al., 2009). As a consequence, this could potentially decrease the level of monosynaptic inhibition which, while functioning primarily as a protective mechanism (as GTO Ib-afferent interneurons inhibit intrafusal fibre impulses if the MTU is overloaded (Moore, 2007, Gabriel et al., 2006, Chalmers, 2002b, Lin et al., 1997, Ovalie, 1987)) can inadvertently destabilise the joint having the adverse effect (Wilson and Flanagan, 2008, Lephart, 2000, Jaric et al., 1995). This also infers that G1 and G2 as late maturers should have had a greater scope for adaptation to the neural stimulus presented by DT. This could therefore have potentially contributed to G1 superior enhancement in DJL performance and dose-response to individualised DT during a period whereby temporary disruptions in neuromuscular coordination have been reported previously (Quatman-Yates et al., 2012, Philippaerts et al., 2006). However, this can only be surmised from this investigations evidence and remains to be elucidated.

Despite having a marginally superior FPMS score (Figure 4.1), G1 pre DT DJL, YBT and SLST measures were inferior compared to G2 advocating they were at greater risk of injury. As G1 were only 0.22 ± 0.54 years post-APHV (Table 3.1) their pre DT intervention scores could be symptomatic of an innate reliance on reactive feedback afferent pathways often observed during circa-pubescence (Lloyd et al., 2011b, Lazaridis et al., 2010, Oliver and Smith, 2010, Grosset et al., 2007, Croce et al., 2004, Lambertz et al., 2003). This notion is supported by G1 YBT sway velocity which was indicative of compensatory postural adjustments. Although reactive feedback is associated with monosynaptic inhibition (Lazaridis et al., 2010, Oliver and Smith, 2010, Grosset et al., 2007, Moore, 2007, Gabriel et al., 2006,

Croce et al., 2004, Lambertz et al., 2003, Chalmers, 2002b), the efficacy of correcting motor control is dependent on the ability to challenge afferent feedback (Comerford and Mottram, 2001a). The sole purpose of DT is to be able to cognitively dissociate movements (Mottram and Comerford, 2008) which reinforces why a superior dose-response following individualised DT was observed for G1. Furthermore, cognitive control has been repeatedly identified as a pathological factor of injury (Verrelst et al., 2014, Verrelst et al., 2013, Leetun et al., 2004, Ireland et al., 2003, Fredericson et al., 2000, Bendjaballah et al., 1997). Conversely, Lloyd et al., (2012) Youth Physical Development model advocates the implementation of specific training to promote feed-forward neural pathways, taking advantage of innate post-pubescent neurophysiological adaptations. The nature of DT however predominantly enhances motor control via afferent feedback which could have potentially attenuated G2 training adaption, compounded by its generic prescription. Furthermore, increased androgen receptor levels post-APHV have been directly associated with morphological adaptations in connective tissue (Fragala et al., 2011, Boisseau and Delamarche, 2000). While a neural stimulus should always be presented, hypertrophy based training is advocated to develop MTU and ligament strength which would attenuate monosynaptic inhibition (Lambertz et al., 2003). Consequently, this would promote feed-forward anticipatory postural adjustments essential to sport-specific motor skills that can contribute to academy footballers' risk of non-contact injury (dos Santos et al., 2014, Lloyd et al., 2012b, Barone et al., 2011, Fragala et al., 2011, Matsuda et al., 2008, Kibler et al., 2006, Ebenbichler et al., 2001, Radebold et al., 2001, Voight et al., 1998). While it can only be surmised from this study's findings the stimulus presented by individualised DT could have been favourable for G1 whereas generic DT could have been unsuitable for G2.

5.4 Implications of Enhancing Motor Control in Elite Male Academy Footballers

Where previous independent remedial motor control programmes have been unsuccessful, individualised DT indicates that it can have a positive dose-response. As DJL, YBT and SLST indicated greater effects than the FPMS this suggests that they are more appropriate surrogate markers of DT efficacy and motor control. It is conceivable that they indicated greater effects as they predominantly focussed on LPHC and lower limb control, whereas the FPMS comprised upper limb assessments which could have had an impact on composite score. However, the kinetic variables measured for all tests assessed lower limb function only. As enhanced lower limb function is governed by proximal control of the LPHC through reciprocal sensory pathways (dos Santos et al., 2014, Hides et al., 2012, Kiers et al., 2012, Kibler et al., 2006, Konin et al., 2003, Devlin, 2000, Hodges and Richardson, 1997, Hodges and Richardson, 1996), G1 and G2 recorded kinetic measures support the proposal that surrogate markers are more appropriate for monitoring DT efficacy. These findings also reinforce Wingfield et al., (2004) and Chalmers et al., (2002a) meta-analysis evidence that the validity of screening select physical competencies as predictors of injury remains equivocal. However, despite the FPMS apparent weaker sensitivity to G1 and G2 changes in motor control the FPMS directed the DT prescription. It therefore remains to be elucidated whether the proposed surrogate markers could direct DT and subsequently have an equal or greater dose-response. However, due to no empirical evidence supporting the FPMS this cannot be ascertained.

In chronological age groups of ≥14 years increases in injury incidence have been consistently reported in elite academy footballers (Cloke et al., 2012, Deehan et al., 2007, Merron et al., 2006, Emery et al., 2005, Malina et al., 2004, Price et al., 2004, Volpi et al., 2003, Peterson et al., 2000, Schmikli and Bol, 1995), with the lower extremity accounting for 90% of non-contact injuries (Ekstrand et al., 2011, Hagglund et al., 2009, Price et al., 2004). These increases could be attributed to the current English EPPP as training and competition increases linearly with chronological age irrespective of biological development (Rumpf and Cronin, 2012). However, there were no significant differences between G1 and G2 match time and frequency throughout the DT intervention or 2013/14 season (Table 4.1). Therefore, G1 post individualised DT evidence advocates injury risk can be attenuated through enhanced motor control. G1 individualised DT enhanced LPHC control superiorly to G2 generic DT, for which there is a plethora of pathological evidence of injury indicating impaired LPHC synergies causes greater uncontrolled displacements (Verrelst et al., 2014, Wilkerson et al., 2012, Chuter and de Jonge, 2012, Willems et al., 2006, Willson et al., 2006, Willson et al., 2005, Niemuth et al., 2005, Leetun et al., 2004, Ireland et al., 2003). By reducing the potential for compensatory kinematics to develop, overload and/or overuse injuries through early onset of neuromuscular fatigue can be attenuated (Mersmann et al., 2014, Parchmann and McBride, 2011, Chorba et al., 2010). G1 greater dose-response to individualised DT indicates that it could be an effective facet of training for elite academy footballers competing under the current English EPPP. Moreover, G2 failure to improve by the same magnitudes as G1 despite a greater DT frequency also means that one individualised DT per week over eight weeks would allow time for complementary training. The importance of this was evidenced by Filipa et al., (2010) whereby

superior magnitudes of change were found following eight weeks multifaceted Integrative Neuromuscular Control Training compared to this study's independent DT intervention.

While G1 CoP measures were indicative of compensatory postural adjustment afferent feedback pathways, LPHC control improved indicating a positive change in motor pattern. Moreover, enhanced compensatory postural adjustments set the foundation for developing anticipatory postural adjustments as enhanced intermuscular coordination decreases the electromechanical delay of resisting forces (Hides et al., 2012, Kibler et al., 2006, Anderson and Behm, 2005, Konin et al., 2003, Behm et al., 2002, Lloyd, 2001, Devlin, 2000, Hodges and Richardson, 1997, Hodges and Richardson, 1996, Kearney and Hunter, 1990). Subsequently, this would elicit an earlier onset and change in activation pattern increasing proximal stability of LPHC allowing enhanced distal mobility and effective attenuation of GRF (Kibler et al., 2006, Ebenbichler et al., 2001, Radebold et al., 2001, Kibler, 1993). As a consequence, G1 onset of neuromuscular fatigue should be attenuated. This is central to motor control training's ability to reduce injury risk as the manifestation of non-contact injuries in elite academy populations has been attributed to this (Mersmann et al., 2014, Meylan et al., 2010, Hawkins and Metheny, 2001, Devlin, 2000). This would elucidate G1 evidenced attenuation of PVGRF and increased TTS through enhanced LPHC control. In addition, this could increase the GRF transferred from the stance to the non-standing leg. This could increase angular velocity, and therefore impulse, reducing the force required to overcome inertia when executing football specific skills (Strauss et al., 2012, Orloff et al., 2008, Harrison and Mannering, 2006, Barfield et al., 2002, Barfield, 1998). Although this is beyond the

realms of this study, this would have significant implications for youth footballers because of the inherent concomitant increased risks of injury and somatic growth, particularly during circa-pubescence (Mersmann et al., 2014, Meylan et al., 2014, Meylan et al., 2010, Beunen and Malina, 2008, Malina et al., 2004, Hawkins and Metheny, 2001).

6.0 Conclusion

6.1 Practical Applications

To enhance motor control DT needs to be individualised to effectively challenge an individual's cognitive processes and afferent feedback. G1 positive response to a low dose of individualised DT also provides practitioners with an effective motor control retraining strategy that can be easily integrated into two traditional four week mesocycle training blocks throughout a season. Moreover, G1 positive dose-response has significant application for elite academy footballers. As G1 assessments of neuromuscular control all improved following only one DT per week over eight weeks this advocates that the overall training workloads of the present EPPP for each age group could be reduced. Consequently, this could attenuate the reported concomitant increase between chronological age group and injury incidence. A high percentage of which are lower extremity non-contact injuries frequently attributed to poor cognitive control and early onset of neuromuscular fatigue. The impact of this study's findings are further accentuated by the fact that only the non-dominant kicking leg of elite academy footballers' were assessed. Therefore, the scope for any

adaptation in motor control would have already been reduced. G1 observed changes in this reduced window of opportunity infer that individualised DT could attenuate potential compensatory synergistic dominant motor skills, previously evidenced to be induced by football training and competition exposure. Although G2 findings indicated that the stimulus presented by generic DT was inadequate, it can only be surmised to what extent G1 and G2 inherent growth and maturational differences affected this study's outcome. Likewise, prospective studies should aim to elucidate the dose-response threshold of individualised DT and its effectiveness in the absence of the FPMS directed prescription. Nonetheless, the main findings of this study advocate that a lower dose of DT is sufficient for improving neuromuscular control provided training is individualised, and is superior to generically prescribed DT. In addition, as SLST, YBT and DJL indicated greater effects than the FPMS and have all been proposed to predict injury through reduced motor control, they could be a more suitable surrogate marker for assessing the effectiveness of DT.

7.0 References

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Appendices

Appendix 1.0: The Foundation Performance Matrix Screen© Guidelines

Appendix 2.0: Participant Information Letter and Informed Consent Form

Appendix 2.1: Parent Information Letter and Informed Consent Form

Appendix 3.0: Example SPSS Output: One-Way Repeated Measures ANOVA and Paired Samples T-Test (Group Foundation Performance Matrix Screen Scores)

Appendix 2.0

Dear Participant

I would like to invite you to volunteer to take part in a research study on behalf of Bristol City Football Club. The study will measure the effects of 8 weeks of the 'motor control training' you already perform during academy training. This will be measured during Bristol City FC Foundation Performance Matrix (FPM) injury screening plus three other measures testing your balance and movement.

This study aims to help Bristol City FC to:

Gain a greater understanding of the mechanisms involved in injury occurrence.

Improve the type of strength training you perform to lower your likelihood of injury.

Improve your athletic performance.

THING YOU NEED TO KNOW IF YOU WOULD LIKE TO TAKE PART:

1. **You will only be included with your signed permission.**
2. **You will need to sign a health questionnaire to make sure you are suitable to participate in the study.**
3. **If under 18 years of age, a separate consent will need to be signed by your parent/ guardian.**

information please contact Gary Davenport, me (E: robertburge@connect.gloas.ac.uk) or my supervisors Dr Jonathan Hughes (E: jhughes1@glos.ac.uk) or Professor Mark De Ste Croix (E: destecroix@glos.ac.uk) on the email details provided. The University of Gloucestershire research ethics subcommittee has approved this study. If you have any concerns please contact Dr Malcolm MacLean, chair of the subcommittee at the University of Gloucestershire, on the details provided (Email: mmaclean@glos.ac.uk, Tel: 01242 715158). Dr MacLean has no direct involvement in the study.

Yours sincerely

Robert Burge

UNIVERSITY OF GLOUCESTERSHIRE
at CHELTENHAM and GLOUCESTER

INFORMATION SHEET: WHAT TESTING INVOLVES

On the morning of each testing YOU will need to:

1. Wear briefs/ cycle shorts or close-fitting undergarments under your shorts;

(When you arrive at training)

2. Take off all upper body clothing when performing the upper body FPM screen movements. This will allow select joints of the upper body to be observed during assessment.

What YOU will be required to perform at Bristol City FC academy training:

1. U15/ U16 age groups will take part in one dissociation training session per week.
2. U17/ U18 age groups will take part in three dissociation training sessions per week.
3. Each session will last no longer than 60 minutes.
4. Gary Davenport and/ or Steve Taylor will supervise all of your training.
5. All training will take place during Bristol City FC academy training.

Two practice sessions will be provided. Correct exercise technique will be taught and any questions about the study answered. The program duration has been set as an appropriate time for any positive change to occur.

What YOU need to know about volunteering for this study:

- As a volunteer you have the right to withdraw from the study at any point without consequence.
- All of your individual data will remain anonymous.
- Data will be kept for a minimum of five years. After five years, all data will be destroyed.
- Bristol City FC sports science and medical staff will be made aware of the study's outcome. They will not have access to your individual data to prevent any bias for your future selection.
- For coaching feedback video analysis will be employed by the researcher, Gary Davenport or Steve Taylor. All video footage will be deleted within 24 hours.
- Continue participating in all physical education and sports throughout the duration of the study.
- This study will be published as the researcher's academic work. A publication may be made available to students in the University of Gloucestershire library and in the public domain
    - **You will not be identifiable by name**.
    - **Your data will be used for research purposes only**.

Participant Informed Consent Form

Title of Project	**The Dose-Response Effects of Dissociation Training on Measures of Neuromuscular Control during Performance Screening in Male Youth Footballers**

Principle investigators	Robert Burge Sports Strength and Conditioning MSc by Research student & Bristol City FC research scholar Faculty of Applied Sciences, University of Gloucestershire, Oxstalls Campus, Oxstalls Lane, Gloucester, GL2 9HW robertburge@connect.glos.ac.uk

I understand what I have been asked to participate in a research study?	Yes	No
I have read and received a copy of the information letter?	Yes	No
I understand the benefits and risks involved in taking part in this research study?	Yes	No
I understand that I am free to contact the researcher to ask questions and discuss this study?	Yes	No
I understand that I am free to not volunteer, or to withdraw from the study at any time, without consequence and that my information will be deleted at my request?	Yes	No
I understand that the researcher will keep my data confidential? I understand who will have access to your information?	Yes	No

I wish to take part in this research study:

Participant Printed Name: _____

Participant Signature: _____

Date: _____

Preferred contact number: _____

Email: _____

Appendix 2.1

Mr Robert Burge
Sports Strength & Conditioning
Faculty of Applied Sciences
Oxstalls Campus
Oxstalls Lane
Longlevens
GLOUCESTER GL2 9HW
robertburge@.connect.glos.ac.uk

22nd January 2014

Dear Parent/ Guardian

The Dose-Response Effects of Dissociation Training on Measures of Neuromuscular Control during Performance Screening in Male Youth Footballers

I am a postgraduate MSc by Research Sports Strength and Conditioning student and research scholar for Bristol City Football Club. I would like to invite your son to take part in a research study. They will only be included with your permission. The purpose of the study is to measure the effects of 8 weeks dissociation training on four measures of balance and dynamic postural control. (Dissociation training is a form of bodyweight corrective movement training to improve muscle coordination. Its aim is to lower the risk of injury by improving identified uncontrolled movements). This will be measured during Bristol City FC injury screening.

The researcher and Bristol City FC academy head of sport science (Gary Davenport) will be present at all times during testing. The researcher is CRB certified to work with youth populations. Participants will be required to wear briefs or close-fitting undergarments under their shorts. Participants will be asked to perform the screening movements without a shirt on. This will allow for observational assessments of select upper body joints. Participants will be asked to fill out a health questionnaire to make sure they are suitable to participate in the study. Participants aged 15 to 16 years will take part in one dissociation training session per week. Participants aged 17 to 18 years will take part in three dissociation training sessions per week. Each session will last no longer than 60 minutes. All sessions will be supervised by Bristol City FC head of sport science and/ or head of physiotherapy. All training will take place during Bristol City FC academy training. Two practice sessions will be provided. Correct exercise technique will be taught and any questions about the study answered. The programme

duration has been set as an appropriate time for any positive change to occur. This study poses a very low risk of injury. As a volunteer, your son reserves the right to withdraw from the study at any point without consequence. All individual data will remain anonymous. Data will only be stored on a password secured laptop locked in the researcher's supervisor's office. Data will be kept for a minimum of five years. After five years, all data will be destroyed.

Bristol City FC sports science and medical staff will be made aware of the study's outcome. They will not have access to individual participant data. This will eliminate any bias in future selection. The results from this study will be presented in my academic thesis. No participant will be identifiable by name. All data will be used for research purposes only. A publication may be made available to students in the University library and in the public domain. For coaching feedback video and photographic analysis will be employed. This will be administered by the researcher or one of Bristol City FC coaching staff. For data protection, any memory card containing images or video footage will be stored in a locked draw in the researcher's supervisor's office. Continued participation in all physical education throughout the duration of the study is encouraged. Participation in this study could help coaches to: Gain a greater understanding of the mechanisms involved in injury occurrence; assist the prescription of strength training to lower the likelihood of injury; and improve athletic performance. There are very low risks associated with taking part in this research.

The University of Gloucestershire faculty research ethics panel has approved this study. Please contact Dr Malcolm MacLean, chair of the research ethics subcommittee for the Faculty of Applied Sciences at the University of Gloucestershire, by email or phone if you have any concerns (Email: mmaclean@glos.ac.uk, Tel: 01242 715158). Dr MacLean has no direct involvement in the study. If you wish for any further information please contact me on my email details provided. Alternatively, please contact my supervisors Dr Jonathan Hughes (E: jhughes1@glos.ac.uk) or Professor Mark De Ste Croix (E: destecroix@glos.ac.uk) on the email details provided.

Yours sincerely

Robert Burge

UNIVERSITY OF GLOUCESTERSHIRE
at CHELTENHAM and GLOUCESTER

Parent/ Guardian Informed Consent Form

Title of Project	**The Dose-Response Effects of Dissociation Training on Measures of Neuromuscular Control during Performance Screening in Male Youth Footballers**

Principle investigators	Robert Burge Sports Strength and Conditioning MSc by Research student & Bristol City FC research scholar Faculty of Applied Sciences, University of Gloucestershire, Oxstalls Campus, Oxstalls Lane, Gloucester, GL2 9HW robertburge@connect.glos.ac.uk

Do you understand that I have asked you to participate in a research study?	Yes	No
Have you read and received a copy of the attached information letter?	Yes	No
Do you understand the benefits and risks involved in taking part in this research study?	Yes	No
Do you understand that you are free to contact the researcher to take the opportunity to ask questions and discuss this study?	Yes	No
Do you understand that you are free to refuse participation, or to withdraw from the study at any time, without consequence and that your information will be withdrawn at your request?	Yes	No
Do you understand that I will keep your data confidential? Do you understand who will have access to your information?	Yes	No

I wish to take part in this study:

Participant Printed Name: _____

Participant Signature: _____

Parent/ Guardian Printed Name: _____

Parent/ Guardian Signature: _____

Date: _____

Preferred contact number: _____

Email: _____

Appendix 3.0

```
GLM PreFPMSscore PostFPMSscore BY Group
  /WSFACTOR=Time 2 Polynomial
  /METHOD=SSTYPE(3)
  /SAVE=SRESID
  /PLOT=PROFILE(Time*Group)
  /EMMEANS=TABLES(Group) COMPARE ADJ(BONFERRONI)
  /EMMEANS=TABLES(Time) COMPARE ADJ(BONFERRONI)
  /EMMEANS=TABLES(Group*Time)
  /PRINT=DESCRIPTIVE ETASQ
  /CRITERIA=ALPHA(.05)
  /WSDESIGN=Time
  /DESIGN=Group.
```

General Linear Model

Within-Subjects Factors

Measure: MEASURE_1

Time	Dependent Variable
1	PreFPMSscore
2	PostFPMSscore

Between-Subjects Factors

		N
Group	1.00	6
	2.00	8

Descriptive Statistics

	Group	Mean	Std. Deviation	N
PreFPMSscore	1.00	26.3333	4.08248	6
	2.00	25.0000	6.18755	8
	Total	25.5714	5.24352	14
PostFPMSscore	1.00	27.0000	4.04969	6
	2.00	24.2500	5.54849	8
	Total	25.4286	4.98790	14

Multivariate Tests[a]

Effect		Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared
Time	Pillai's Trace	.002	.019[b]	1.000	12.000	.892	.002
	Wilks' Lambda	.998	.019[b]	1.000	12.000	.892	.002
	Hotelling's Trace	.002	.019[b]	1.000	12.000	.892	.002
	Roy's Largest Root	.002	.019[b]	1.000	12.000	.892	.002
Time * Group	Pillai's Trace	.317	5.567[b]	1.000	12.000	.036	.317
	Wilks' Lambda	.683	5.567[b]	1.000	12.000	.036	.317
	Hotelling's Trace	.464	5.567[b]	1.000	12.000	.036	.317
	Roy's Largest Root	.464	5.567[b]	1.000	12.000	.036	.317

a. Design: Intercept + Group
Within Subjects Design: Time

b. Exact statistic

Mauchly's Test of Sphericity[a]

Measure: MEASURE_1

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Time	1.000	.000	0	.	1.000	1.000	1.000

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept + Group
Within Subjects Design: Time

b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	.012	1	.012	.019	.892	.002
	Greenhouse-Geisser	.012	1.000	.012	.019	.892	.002
	Huynh-Feldt	.012	1.000	.012	.019	.892	.002
	Lower-bound	.012	1.000	.012	.019	.892	.002
Time * Group	Sphericity Assumed	3.440	1	3.440	5.567	.036	.317
	Greenhouse-Geisser	3.440	1.000	3.440	5.567	.036	.317
	Huynh-Feldt	3.440	1.000	3.440	5.567	.036	.317
	Lower-bound	3.440	1.000	3.440	5.567	.036	.317
Error(Time)	Sphericity Assumed	7.417	12	.618			
	Greenhouse-Geisser	7.417	12.000	.618			
	Huynh-Feldt	7.417	12.000	.618			
	Lower-bound	7.417	12.000	.618			

Tests of Within-Subjects Contrasts

Measure: MEASURE_1

Source	Time	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Linear	.012	1	.012	.019	.892	.002
Time * Group	Linear	3.440	1	3.440	5.567	.036	.317
Error(Time)	Linear	7.417	12	.618			

Tests of Between-Subjects Effects

Measure: MEASURE_1

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	18040.012	1	18040.012	337.503	.000	.966
Group	28.583	1	28.583	.535	.479	.043
Error	641.417	12	53.451			

Estimated Marginal Means

1. Group

Estimates

Measure: MEASURE_1

Group	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1.00	26.667	2.111	22.068	31.265
2.00	24.625	1.828	20.643	28.607

Pairwise Comparisons

Measure: MEASURE_1

(I) Group	(J) Group	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1.00	2.00	2.042	2.792	.479	-4.041	8.125
2.00	1.00	-2.042	2.792	.479	-8.125	4.041

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

Univariate Tests

Measure: MEASURE_1

	Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Contrast	14.292	1	14.292	.535	.479	.043
Error	320.708	12	26.726			

The F tests the effect of Group. This test is based on the linearly independent pairwise comparisons among the estimated marginal means.

2. Time

Estimates

Measure: MEASURE_1

Time	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	25.667	1.461	22.483	28.850
2	25.625	1.345	22.696	28.554

Pairwise Comparisons

Measure: MEASURE_1

(I) Time	(J) Time	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.042	.300	.892	-.612	.696
2	1	-.042	.300	.892	-.696	.612

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

Multivariate Tests

	Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared
Pillai's trace	.002	.019[a]	1.000	12.000	.892	.002
Wilks' lambda	.998	.019[a]	1.000	12.000	.892	.002
Hotelling's trace	.002	.019[a]	1.000	12.000	.892	.002
Roy's largest root	.002	.019[a]	1.000	12.000	.892	.002

Each F tests the multivariate effect of Time. These tests are based on the linearly independent pairwise comparisons among the estimated marginal means.

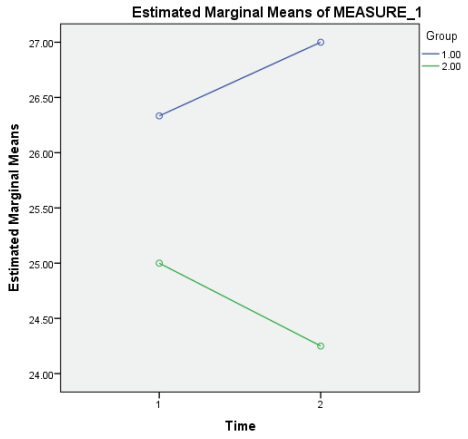
a. Exact statistic

3. Group * Time

Measure: MEASURE_1

Group	Time	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
1.00	1	26.333	2.209	21.520	31.146
	2	27.000	2.033	22.571	31.429
2.00	1	25.000	1.913	20.832	29.168
	2	24.250	1.760	20.414	28.086

Profile Plots




```
T-TEST PAIRS=PreFPMSscoreG2 PreFPMSscoreG1 PreFPM92SwayVelocityG2 PreFPM92SwayVelocityG1 WITH
PostFPMSscoreG2 PostFPMSscoreG1 PostFPM92SwayVelocityG2 PostFPM92SwayVelocityG1 (PAIRED)
  /CRITERIA=CI(.9500)
  /MISSING=ANALYSIS.
```

T-Test

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	PreFPMSscoreG2	25.0000	8	6.18755	2.18763
	PostFPMSscoreG2	24.2500	8	5.54849	1.96169
Pair 2	PreFPMSscoreG1	26.3333	6	4.08248	1.66667
	PostFPMSscoreG1	27.0000	6	4.04969	1.65328
Pair 3	PreFPM92SwayVelocityG2	9.9463	8	1.79243	.63372
	PostFPM92SwayVelocityG2	8.5163	8	4.22097	1.49234
Pair 4	PreFPM92SwayVelocityG1	8.7467	6	2.41889	.98751
	PostFPM92SwayVelocityG1	11.7033	6	2.32977	.95112

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	PreFPMSscoreG2 & PostFPMSscoreG2	8	.990	.000
Pair 2	PreFPMSscoreG1 & PostFPMSscoreG1	6	.956	.003
Pair 3	PreFPM92SwayVelocityG2 & PostFPM92SwayVelocityG2	8	.295	.479
Pair 4	PreFPM92SwayVelocityG1 & PostFPM92SwayVelocityG1	6	.134	.800

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	PreFPMSscoreG2 - PostFPMSscoreG2	.75000	1.03510	.36596	-.11536	1.61536	2.049	7	.080
Pair 2	PreFPMSscoreG1 - PostFPMSscoreG1	-.66667	1.21106	.49441	-1.93760	.60426	-1.348	5	.235
Pair 3	PreFPM92SwayVelocityG2 - PostFPM92SwayVelocityG2	1.43000	4.07078	1.43924	-1.97326	4.83326	.994	7	.354
Pair 4	PreFPM92SwayVelocityG1 - PostFPM92SwayVelocityG1	-2.95667	3.12479	1.27569	-6.23593	.32260	-2.318	5	.068