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**Title:** The influence of age and maturation on trajectories of stretch shortening cycle capability in male youth team sports

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**Abstract:**

**Purpose:** To examine the the influence of growth and maturation in the trajectory of stretch shortening cycle (SSC) capability. **Method:** Using a mixed longitudinal design absolute and relative leg stiffness, and reactive strength index (RSI) were measured three times over a three-year period in 44 youth team sport players. Maturation was determined as maturity-offset and included within the Bayesian inference analysis as a covariate alongside chronological age. **Results:** Irrespective of age and maturation there was no change in absolute leg stiffness, however relative leg stiffness decreased over time. Maturation and age reduced this decline, but the decline remained significant ( $BF_{10} = 5097$ , model averaged  $R^2 = 0.61$ ). RSI increased over time and more so in older more mature youth players ( $BF_{10} = 9.29e^8$ , model averaged  $R^2 = 0.657$ ). **Conclusion:** In youth players who are at/post peak height velocity, relative leg stiffness appears to decline, which could have an impact on both performance and injury risk. However, RSI increases during this period, and these data reinforce that leg stiffness and RSI reflect different components of SSC capability. Practitioners should consider these differences when planning training to maximise SSC capability during growth and maturation in athletes on the developmental performance pathway.

**Introduction:**

The stretch-shortening cycle (SSC) is defined as a muscle action that involves preactivation of the muscle prior to ground contact, a fast-eccentric action, and a rapid transition between the eccentric and concentric phases (10). Throughout both soccer and basketball matches, players routinely utilize SSC actions, especially in high-intensity movements that require

high levels of rate of force development, such as maximal velocity running and jumping. Leg stiffness is commonly used as a measure to characterize SSC function, and represents the ratio between peak vertical ground reaction forces and peak center of mass displacement during ground contact (22). Comprising stiffness regulation around all the joints of the lower limb, this metric functionally represents how individuals have to control multi-joint movements during SSC exercise (10) and is regulated by feed-forward and feedback neural mechanisms, which appear to develop from childhood into adulthood (20,29). Low levels of leg stiffness compromise physical performance and are well related to sprinting and jumping performance (22,30). Squat jump and countermovement jumps have previously been used as field-based tests to determine SSC capability and the percentage difference between both measures attributed to prestretch augmentation. However, these movements do not use a stretch reflex and therefore do not appropriately reflect SSC function (10). Given that measurements of fast SSC require ground contact times of <250ms these methods do not permit investigation of fast SSC (37). Rebound based testing elicits preactivation and a stretch reflex in the lower limbs which are the fundamental prerequisites of SSC behaviour (10). Field based measures of leg stiffness and reactive strength index (RSI) are rebound tests using a spring mass model approach which have been shown to be reliable measures of SSC capability in youth populations (4,17)

Understanding changes in stiffness during growth and maturation are essential in helping to develop appropriate performance enhancing and injury management strategies (1, 26). Knee joint stability and protection against injury relies on adequate feedback and feed-forward systems to improve muscular stiffness during functional tasks (35,39). Lower limb (leg) stiffness points to an ability to generate strength and resist to deformation resulting from movement including a direct transition from eccentric to concentric muscle contraction (31). An increase in this ability leads to a lesser probability of excessive load of the knee passive

structures such as the ACL (8). Assessment of lower limb stiffness provides significant information relating to neuromuscular pre-activation and neuromuscular feed-forward control. It is hypothesized that increases in body mass as a result of puberty will lead to increases in absolute leg stiffness in order to maintain true spring-mass model behaviour during ground contact (7), however, owing to the limited published literature for paediatric leg stiffness measures, whether the same trend exists for relative stiffness is unclear.

A number of studies examining leg stiffness in youth athletes are available, with many focusing on the changes in SSC capability after performance related tasks, such as simulated and competitive match play (4,5,15,16,28). However, few studies have explored the innate development in SSC capability throughout childhood and adolescence. In a recent review article, Radnor et al. (32) suggest that SSC performance increases non-linearly with age during various hopping and jumping tasks. This assumption is based on limited cross-sectional data, and the author acknowledge this by concluding that longitudinal studies that include measures of growth and maturation are needed to provide evidence for the natural development of SSC capability (32). From those studies that are available Oliver and Smith (29) found significantly higher leg stiffness in adult males compared to pre-pubescent boys during submaximal hopping at high hopping frequencies. In a small cross-sectional sample ( $n = 32$ ) Lloyd et al. (20) found pre-pubescent boys (9–12 years) to have lower leg stiffness compared to adolescent boys aged 15 years and adults. However, in a more comprehensive cross-sectional study ( $n = 250$ ) the same authors showed that relative leg stiffness increased between 7-10y, declined between 10-12y, increased again between 12-13y and then remained similar until 16y. This variable change across chronological age groups leads the authors to the conclusion that the development of leg stiffness is unique within children. Laffaye et al. (14) also demonstrated that relative stiffness increased by only a small amount between 11-16y (+17%) but then significantly increased between 17-20y (+32%). Indeed, their data also shows a decline in relative stiffness in boys

between 11-12y and 13-14y, with the authors concluding that the evolution of leg stiffness does not occur until 15-16 years of age. Lloyd et al. (18,20) tentatively suggest that the negative effects in leg stiffness appear around 12-18 months prior to peak height velocity (PHV) and might reflect a decline in motor performance resulting from the differential growth spurts in leg and trunk length. Longitudinal studies are needed to further reinforce whether such reductions in performance might be attributable to the theory of adolescent awkwardness.

From the point of view of protection against ACL injury, it was also suggested that reactive strength expressed by means of reactive strength index (RSI) is another potential indicator of ACL injury (27,33,37). Youth athletes with a higher RSI should be considered as individuals at a lower injury risk or as more robust athletes (which may be demonstrated from fewer days of absence from training) as shown in youth alpine ski racers (25). Studies dealing with the influence of chronological age on RSI point to a gradual improvement during adolescence (18,19). A cross-sectional study in school children reported that the smallest worthwhile changes between age groups indicated a naturally occurring accelerated adaptation in RSI (18). Laffaye et al. (14) showed no change in RSI from 11-12 to 13-14y but then a significant increase from 13-14 to 15-16y in untrained boys. However, to date there are no longitudinal studies exploring changes in RSI when age and maturation are taken into account. Previous authors have indicated that RSI is more indicative of slow-SSC development and leg stiffness reflects the trend for fast-SSC development and thus it is important to measure both components as the age and maturation related changes may be different (18,20).

Current data is also limited by the statistical approach taken to exploring the age and maturation related changes in SSC capability. Bayesian methods have been suggested as an alternative to traditional frequentist statistics for a number of reasons (12). Unlike the frequentist approach (where only a dichotomous response provides information regarding the presence or absence of effects), the Bayesian factor [BF] explores the quantification of the

relative degree of evidence for supporting the null hypothesis vs the alternative hypothesis without being ‘violently biased’ against the null hypothesis (12). Also, in contrast to using a p-value the BF can be meaningfully interpreted even when the data at hand is generated via real world processes outside of ‘true’ experimental control (39). Thus, Bayesian methods permit a more robust exploration of the effects of growth and maturation on SSC capability and may provide us with an alternative understanding of these effects.

Monitoring of SSC capability during adolescence, especially during PHV and for up to two years (at least) following the age of PHV should be a priority given that injury incidence rates are greatest during this period (34). This is also perceived as an important period in the development of performance related outcomes (eg speed, jump height) that might be influenced by ‘adolescent awkwardness’ (32). To the best of our knowledge there are no mixed longitudinal studies focusing on the influence of age and maturation on leg stiffness and RSI in youth athletes. Therefore, the main aim of this study was to determine the effect of chronological age and maturation on changes in SSC capability in male youth male players of team sports.

## **Methods**

### ***Participants***

For the purpose of this mixed-longitudinal study, a group of 89 male youth soccer and basketball players from two chronological competition age groups at the start of the study, U14y [n =44] (age  $13.2 \pm 0.5$ y; maturity offset  $-0.02 \pm 0.68$ y; stature  $1.66 \pm 0.94$ m; body mass  $52.5 \pm 9.7$ kg; training age 6-7y) and U16y [n = 45] (age  $15.1 \pm 0.6$ y; maturity offset  $1.69 \pm 0.71$ y; stature  $1.80 \pm 0.66$ m; body mass  $66.9 \pm 8.8$ kg; training age 8-9y) were recruited. Players were all on an elite developmental pathway and trained five to seven times per week (equating to 6-12hr) and played one competitive match per week (soccer) or two matches every other week (basketball). Training consisted of typical age-related training for youth athletes in an elite pathway: i.e. physical fitness training (mainly strength and

power, speed and agility with and without ball, repeated sprint ability with and without ball), skill-oriented training (technical-tactical training, game-like training), recovery training.

The study was approved by the Institution's ethics committee and conformed to the Declaration of Helsinki regarding the use of human subjects. The criteria for inclusion into the study was absence of serious thigh and knee injuries in the six months prior to the first measurement. All players were fully informed about the aim of the study and the testing procedures that would be employed. Written informed consent to the testing procedures and the use of the data for further research was obtained from the players' parents and the children, as well as verbal assent from all players. Testing occurred over three competitive seasons 2016/17, 2017/18 and 2018/19 and approximately three weeks into each season. The day before testing, the players were not exposed to any high intensity exercises. The final sample size for data analysis was 44 participants (U14y [n=20] and U16y [n = 24]) as these players completed all three years measurements. The reasons for exclusions were non-participation in training sessions due to injury longer than 4 weeks during the observed competitive season, absence during a measurement session, change of club, or release from the club.

### *Anthropometrics*

Chronological age was determined using the date of birth and the date of the testing session at each time point. Biological maturity of the players at each time point was predicted as the offset from PHV using the sex appropriate equation of Mirwald et al. (23). Leg length, tibia length, standing and sitting height measures were obtained using a stadiometer A-226 Anthropometer (Trystom, CR). Body mass was measured using Tanita UM-075 weighing scales (Tanita, Japan). There were statistically significant differences between both age groups for all anthropometric measures as can be seen in Table 1.



Table 1 Descriptive characteristics (mean  $\pm$  standard deviation) of the participant at the beginning of the study (year 0)

	U14	U16
Variable		
▪ Age (y)	13.2 $\pm$ 0.5	15.1 $\pm$ 0.6*
▪ Stature (m)	1.66 $\pm$ 0.94	1.80 $\pm$ 0.66*
▪ Bodymass (kg)	52.5 $\pm$ 9.7	66.9 $\pm$ 8.8*
▪ Maturity offset (y)	-0.02 $\pm$ 0.68	1.69 $\pm$ 0.71*

m: meter; y: year; kg: kilogram; \*:there were inter-age group differences in all descriptive variables

### *Leg stiffness*

Leg stiffness was calculated from contact time data obtained during a sub-maximal bilateral hopping protocol. Coefficient of variation for male youth has been reported to be 9.4% (17). Players performed three sets of a 20 sub-maximal bilateral hopping protocol. For each trial, participants were instructed to perform consecutive hops on a mobile 2-axis force platform PS-2142 (Pasco, Roseville, CA, USA) at a frequency of 2.5 Hz to reflect the typical behaviour of a spring model (14). Hopping frequency was maintained via an audio signal from quartz Wittner metronome (WITTNER, Isny, Germany). Participants were instructed to: a) keep hands on the hips at all times to avoid upper body interference; b) jump and land on the same spot; c) land with legs fully extended and to look forward at a fixed position to aid balance. For data analysis the first 4 hops were discounted and the next 10 consecutive hops closest to the hopping frequency were used. Absolute leg stiffness ( $\text{kN}\cdot\text{m}^{-1}$ ) was calculated using the equation proposed by Dalleau, Belli, Viale, Lacour, and Bourdin (2).

Equation 1 Where  $K_N$  is the leg stiffness,  $M$  is total body mass,  $T_c$  the ground contact time and  $T_f$  the flight time

$$K_N = \frac{M \cdot \pi (T_f + T_c)}{T_c^2 \left( \frac{T_f + T_c}{\pi} - \frac{T_c}{4} \right)}$$

Relative leg stiffness was determined by dividing absolute leg stiffness by body mass and limb length to provide a dimensionless value. This method has been shown to be valid and reliable in youth athletes with a CV of 8-10% and a TEE of 7.5% (4,17).

### *Reactive strength index*

RSI was determined during a 5 maximum hop test which was performed on a mobile contact mat (FITRO Jumper, Fitronic, Bratislava, Slovakia) and the RSI variable was calculated using the equation of Flanagan and Comyns (6). Participants were instructed to maximize jump height and minimize ground contact time (2). The first hop served as a counter movement jump (impetus) and was consequently excluded from analysis, with the 4 remaining hops averaged for analysis of RSI. Players performed three trials (one practice, and two measured trials) wearing trainers with a 2 minute rest between trials. The trial that resulted in the highest value of RSI was used in further analysis. This method has been shown to be valid and reliable in youth athletes with CVs of 13% reported (17).

### **Statistical methods**

Statistical analyses were performed using JASP (Amsterdam, Netherland) software version 0.10. Descriptive statistics including means and standard deviations were calculated for each variable separately by year (year 0 [initial testing], year 1 [intermediate testing] and year 2 [final testing]). Separate Bayesian inference analyses with JZS prior and r scale of 0.354 were conducted to explore the potential effects of the factor year (three levels) on each neuromuscular measure assessed (absolute and relative leg stiffness and RSI). As this is a two-year longitudinal study (data panel approach), neither age nor maturity offset were discretized and added as fixed factors in the Bayesian inference analyses but as covariables. Therefore, all participants were included in each Bayesian ANCOVA carried out. The age and maturity offset were added as covariables. Furthermore, players' ID was added as random factor in each Bayesian analysis. In each of the models generated, the quantification of the relative degree of evidence for supporting the null hypothesis ( $H_0$  = no effect) or alternative hypothesis ( $H_1$  =

relevant effect) was performed by means of the Bayesian factor ( $BF_{10}$ ) (21,24,36). The  $BF_{10}$  was interpreted using the evidence categories suggested by Lee and Wagenmakers:  $< \frac{1}{100}$  = extreme evidence for  $H_0$ , from  $\frac{1}{100}$  to  $< \frac{1}{30}$  = very strong evidence for  $H_0$ , from  $\frac{1}{30}$  to  $< \frac{1}{10}$  = strong evidence for  $H_0$ , from  $\frac{1}{10}$  to  $< \frac{1}{3}$  = moderate evidence for  $H_0$ , from  $\frac{1}{3}$  to  $< 1$  = anecdotal evidence for  $H_0$ , from 1 to 3 = anecdotal evidence for  $H_1$ , from  $> 3$  to 10 = moderate evidence for  $H_1$ , from  $> 10$  to 30 = strong evidence for  $H_1$ , from  $> 30$  to 100 = very strong evidence for  $H_1$ ,  $> 100$  = extreme evidence for  $H_1$ . Only those models that showed at least strong evidence for supporting the alternative hypothesis ( $BF_{10} > 10$ ) with a percental error  $< 10$  were considered robust enough to describe the main effects. Consequently, when the fixed factor year showed at least a strong (ten times higher) evidence for supporting  $H_1$  ( $BF_{10} > 10$ ) and a percental error  $< 10$ , a posterior post hoc analysis was then carried out. In the post hoc analysis, posterior odds were corrected for multiple testing by fixing to 0.5 the prior probability that the null hypothesis holds across all comparisons (38).

## Results

Descriptive statistics (mean  $\pm$  standard deviation) for leg stiffness and RSI are displayed in table 2.

Table 2 Descriptive statistics (mean  $\pm$  standard deviation) for each variable by year.

Variable	Year		
	Year 0 (Initial testing)	Year 1 (Intermediate testing)	Year 2 (Final testing)
▪ Absolute Leg Stiffness (kN)	26.6 $\pm$ 5.4	25.5 $\pm$ 5.0	26.1 $\pm$ 4.6
▪ Relative Leg Stiffness (kN·m <sup>-1</sup> )	36.5 $\pm$ 6.2	34.1 $\pm$ 5.3	32.8 $\pm$ 3.0
▪ RSI	1.49 $\pm$ 0.37	1.68 $\pm$ 0.37	1.78 $\pm$ 0.4

Table 3 shows the adjusted means model from the Bayesian ANCOVA.

Table 3 Model (ANCOVA) averaged posterior statistics for the relative stiffness and reactive strength index variables

Variable	Full model regression equation	Model average R <sup>2</sup>	Adjusted means*		
			Initial testing session	Intermediate testing session	Final testing session
Relative stiffness (kN·m-1)	$34.548 + 1.899y_0 - 0.350y_{1st} - 1.549y_{2nd} - 0.143a + 0.244mt$	0.61 (0.53 to 0.67)	36.4 (35.4 to 37.3)	34.1 (33.3 to 34.8)	33.0 (32.1 to 33.9)
Reactive strength index	$1.652 - 0.045y_0 + 0.016y_{1st} + 0.029y_{2nd} + 0.134a - 0.016mt$	0.66 (0.52 to 0.74)	1.51 (1.43 to 1.58)	1.70 (1.65 to 1.74)	1.79 (1.72 to 1.87)

\*: adjusted means (95% credible intervals) to both covariables (age and maturity offset)

y: year, m: meter, N: Newton, k: kilogram, mt: maturity offset

The results support the null hypothesis that the fixed factor year ( $BF_{10} = 0.659$  [anecdotal evidence]) and the covariables of age ( $BF_{10} = 0.036$  [strong evidence]) and maturation ( $BF_{10} = 0.058$  [strong evidence], intercept = 0.239 [95% credible intervals = -0.793 to 1353]) have neither individual nor collective effects on participants' absolute leg stiffness (figure 1).

The results also reveal that the alternative hypotheses that the fixed factor year ( $BF_{10} = 27295$  [extreme evidence]) and the covariables of age ( $BF_{10} = 491$  [extreme evidence], intercept = -0.142 [95% credible intervals = -1.404 to 0.912]) and maturation ( $BF_{10} = 15.1$  [strong evidence], intercept = 0.239 [95% credible intervals = -0.793 to 1353]) have individual main effects on participants' relative stiffness (more likely than the null hypothesis of no effect). When both covariables (age and maturation) were added to the year model, this decreased the odds for the alternative hypothesis by 5-fold, but they were still contributed significant independent effects ( $BF_{10} = 5097$  [extreme evidence], model averaged  $R^2 = 0.61$ ). The posterior post hoc analysis conducted for the factor year showed that there was only at least strong evidence to support the alternative hypothesis in the paired comparison conducted between years 0 and 2 ( $BF_{10} = 72.4$  [very strong evidence]), whereby at the beginning of the study (year 0) the relative stiffness scores were higher than those two year later (figure 2).

The results also support the alternative hypotheses that the factor year ( $BF_{10} = 1.32e^7$  [extreme evidence]) and the covariates age ( $BF_{10} = 1.93e^{10}$  [extreme evidence] intercept = 0.134 [95% credible intervals = 0.053 to 0.214]) and maturation ( $BF_{10} = 3.85e^6$  [extreme evidence], intercept = -0.019 [95% credible intervals = -0.018 to 0.092]) have individual main effects on RSI. When the covariables age and maturation were added to the model for the factor year, this increased the odds for the alternative hypothesis by 200-fold ( $BF_{10} = 9.29e^8$  [extreme evidence], model averaged  $R^2 = 0.657$ ). The posterior post hoc analysis indicates at least strong evidence for supporting the alternative hypothesis for the paired comparison between years 0

and 1 ( $BF_{10} = 260.9$  [extreme evidence]), whereby there was a significant increase in the RSI scores between years 0-2 (figure 3).

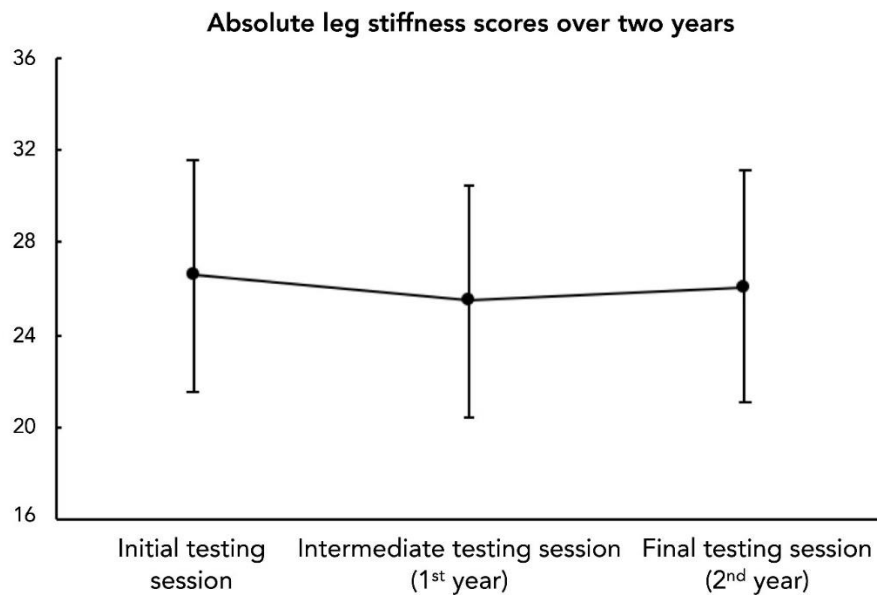


Figure 1 Absolute leg stiffness by year

\* Significant difference to initial testing session

Points are mean values and error bars represent the 95% credible intervals

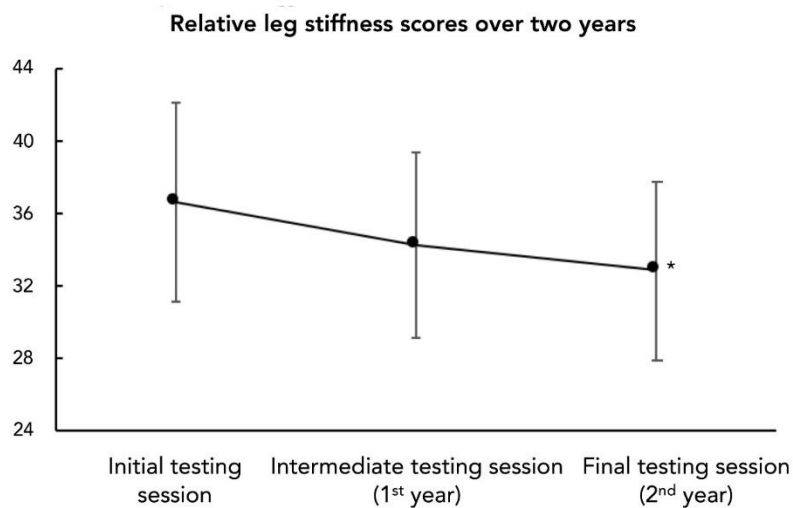


Figure 2 Relative leg stiffness by year

\* Significant difference to initial testing session

Points are mean values and error bars represent the 95% credible intervals

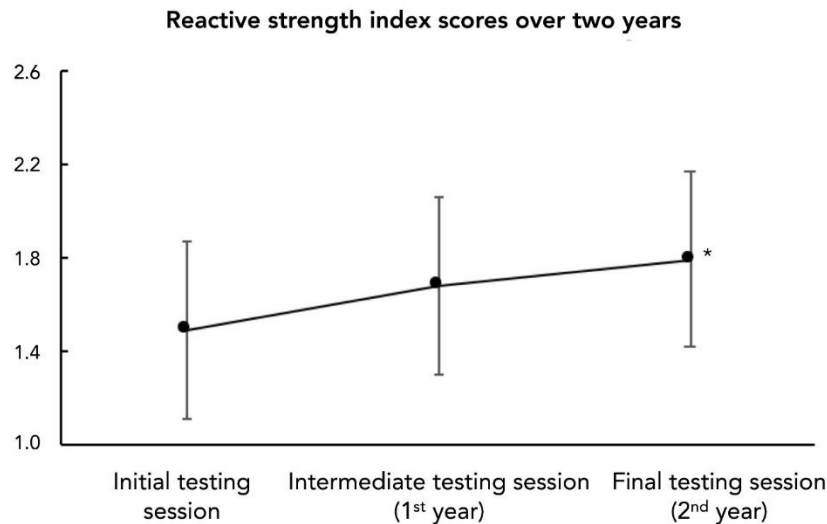


Figure 3 Reactive Strength Index by year

\* Significant difference to initial testing session

Points are mean values and error bars represent the 95% credible intervals

## Discussion

To our knowledge this is the first mixed longitudinal study exploring changes in SSC capability determined by leg stiffness and RSI, whilst exploring the effects of chronological age and maturation. This also appears to be the first study to employ a Bayesian analysis to mixed longitudinal data when exploring the age and maturation related changes in SSC capability. Radnor et al. (32) suggest that as children transition towards adulthood they demonstrate a natural improvement in jumping and hopping tasks, which reflects improvements in SSC capability. The increase in RSI in the current study supports the view that SSC capability increases with chronological age. This is somewhat expected given that increases in body mass as a result of puberty lead to increases in absolute leg stiffness in order to maintain true spring-mass model behaviour during ground contact (7,18,19). Indeed, previous studies have shown a relatively linear increase in absolute stiffness with chronological age from cross-sectional studies (18,20). However, our longitudinal data did not show an increase in absolute leg stiffness with age and relative leg stiffness declined with age. It is possible that the cross-sectional design of previous studies may have clouded our

understanding of the changes in fast SSC during growth and maturation, especially in those individuals involved in an elite developmental pathway.

The data from the current longitudinal study, employing a Bayesian analysis, indicated that irrespective of age and maturation that absolute leg stiffness did not change ( $BF_{10} = 0.659$  anecdotal evidence) and that relative leg stiffness decreased at each testing year ( $BF_{10} = 27295$  extreme evidence). Although the decline in relative stiffness was reduced 5 fold when both age and maturation were included into the analysis there was still extreme evidence that relative leg stiffness declines at each time point. These data are in contrast to the previously reported literature that suggests an increase in relative leg stiffness with age, albeit non-linear in fashion (32). Previous studies have suggested that relative leg stiffness is age specific and gradually increases during adolescence because athletes become more reliant on feed-forward mechanisms as they mature (4). However, we found a decline in relative leg stiffness irrespective of age and maturation whereas Lloyd et al. (18,20) report a plateauing of relative leg stiffness from 13-16+y. In contrast Laffaye et al. (14) reported a significant increase in relative leg stiffness from 13-14y to 15-16y, although there is a difference in how leg stiffness was determined with Laffaye et al. (14) using a 5 maximum hop test. It is likely that a 5 maximum hop test does not challenge the fast SSC, which is in contrast to employing a 20 submaximal hopping test. This in part may explain some of the differences in the trajectory of relative stiffness with age between the current study and that of Laffaye et al (14). However, these authors also suggest that there is a threshold in the evolution of leg stiffness at around the age of 15–16 years (14) and the influence of growth and maturation on the values of leg stiffness occur during the circa-pubertal period. It is important to note that Lloyd et al (19,20) demonstrated a steady increase in absolute leg stiffness across all age groups from 7-16y (increase 7-10y and 12-13y) and the reduction in relative stiffness from 10-12y is likely due to disproportionate increases in limb length and changes in body composition. Lloyd et al. (19,20)



did report similar negative changes for relative leg stiffness, however these decrements were in children who were 12-18 months before PHV (approx. 10-12y-old). The participants in the younger age group in the current study were all pre or around PHV at the first testing session and around 2yr post PHV at the last test occasion, and this may account for the differences in the changes in absolute and relative leg stiffness between our study and that of Lloyd et al. (18,20). Another reason why we may have found a difference compared to the studies of Lloyd et al. (18,20) and Laffaye et al. (14) is that they employed a cross-sectional design using untrained school children in their study, opposed to the sporting group used in the current study. Given that there is a suggestion that training may increase leg stiffness over and above changes expected during normal growth and maturation it is surprising that we have found a decline in relative leg stiffness. The findings that relative leg stiffness decline over time, even with chronological age and maturation accounted for, are somewhat difficult to interpret. It may simply be, as reported by Lloyd et al. (18,20) that greater body mass and limb length negatively impact on SSC capability as these occur prior to large increases in muscle mass and fibre type. Radnor et al. (32) noted that the potentially higher proportion in type II muscle fibres that develop during maturation may result in a greater ability to rapidly generate force due to a faster shortening velocity compared to type I fibres. However, previous studies have reported that tendon stiffness increases with age and reaches adult stiffness values around PHV (13). Given that our participants are around PHV at the first testing session suggests they may already have adult like mechanical properties of the tendon. Interestingly Radnor et al. (32) postulate that adolescents may not have the ability to create optimal muscle stiffness through adequate motor unit recruitment or muscle activation strategies, despite having similar tendon stiffness to adults. This may in part explain the reduction in relative stiffness found in our adolescent players. Iga et al. (9) also previously reported that traditional football training in elite youth soccer players creates quadricep dominant athletes which may result in greater

antagonistic co-activation. Aberrant co-contraction neural factors may result in reduced proprioception and less efficient movement, culminating in a less efficient SSC action (32). It may be that traditional training in the soccer and basketball players in the current study may have an impact on the development of fast SSC capability, however this hypothesis requires further research with appropriate control groups and a range of training techniques. The decline in fast SSC capability with maturation is important, given that impairments in the spring mass system result in abnormal loading of musculoskeletal tissue (3). As the musculoskeletal system is relatively immature and still developing during growth and maturation any abnormal loading on the tissue increases the risk of injury. As enhanced joint stiffness augments fundamental joint movement any reduction in that stiffness compromises joint movement and load absorption (3).

To our knowledge this is the first longitudinal study exploring changes in RSI, irrespective of age and maturation, in trained youth. Our findings support those of previous studies that point to a gradual improvement in reactive strength during adolescence, which may in part be due to the development of motor control (18,20). The improvement of RSI ( $BF_{10} = 1.32e^7$  [extreme evidence]), which is increased when both age and maturation are taken into account, may be attributed to the adaptations that occur to the musculo-tendon unit and neuromuscular system. These changes with growth and maturation should induce a positive effect on rapid force producing potential, subsequent enhanced utilisation of the underpinning mechanisms of the SSC, which in turn enhance SSC function. The proposed mechanisms that might explain the increase in RSI with age and maturation observed in the current study include increases in motor-unit recruitment and preactivation, increased tendon stiffness and decreased co-contraction (32). The potential reduction in the agonist-antagonist co-contraction with maturation may be due to a desensitisation of the golgi tendon organs, resulting in greater net force output (32). The difference in the pattern of the natural development of both fast and slow

SSC capability in our cohort further reinforces that leg stiffness and RSI measure different components of SSC capability (4,14).

It is difficult to account for any training effects in the current study as we did not have a control group with which to make comparisons. However, previous studies examining the natural development of SSC capability with age and maturation have shown improvements with age and suggest that training (especially plyometric training) could induce greater improvements in SSC with growth and maturation (32). It is therefore interesting that we did not see any improvements in fast SSC as determined from the leg stiffness task and indeed this declined with both age and maturation. It is difficult to prescribe potential reasons for the decline in relative leg stiffness with age and maturation however it may in part be due to increases in body fat rather than muscle mass as a percentage of overall mass. It is also important to note that the method used to determine maturity offset in the current study (Mirwald equation) has a potential margin of error of 0.5y, although a recent study has indicated that the equation is stable from  $-1$  year through  $+2$  years of PHV (11). It was not the intention of the current study to explore if different methods of determining maturation (eg % predicted adult stature) impact on changes in SSC but future studies employing different techniques are needed.

The current study mixed longitudinal study suggests that previous studies, employing a cross-sectional approach using non-trained subjects, may have clouded our understanding of age and maturation related changes in fast SSC in youth involved in the elite developmental pathway. Our data does however reinforce that the development of RSI and stiffness with age and maturation, in youths who are training, reflect different components of SSC capability. Given that both components appear to follow a different developmental trend it is important for practitioners to use this knowledge to prescribe appropriate fast or slow SSC training during growth and maturation. As plyometric exercise has been shown to increase SSC in youth

populations (32) it may be prudent to include more hopping and bounding activities in players around PHV. It is also important to consider in future longitudinal studies if single sport specialisation may have a dampening effect on fast SSC capability, and the subsequent impact this may have for both jump and sprint performance and injury risk.

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