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ORIGINAL RESEARCH

AHEAD OF THE PAIN: TEMPORAL PRECURSORS TO OSGOOD-SCHLATTER'S DISEASE IN MALE PREMIER LEAGUE ACADEMY FOOTBALLERS – A NESTED CASE-CONTROL STUDY

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ABSTRACT

Introduction: Osgood-Schlatter Disease (OSD) is a growth-related apophyseal injury affecting 7–21% of athletic adolescents. It is commonly linked to repetitive loading of the tibial tuberosity during periods of rapid growth. OSD can have long-term implications on function and quality of life. Despite previous research, it is still unclear which functional, and growth-related factors predispose young athletes to OSD. The aim of this study is to identify functional, growth, and maturation-related factors associated with the development of OSD in male Premier League academy footballers.

Methods: Retrospective data from 2019 to 2024 were analysed from 88 male academy footballers (48 with OSD, 40 controls) from age U12 to U18. OSD onset was clinically diagnosed by academy medical staff. Somatic maturation was estimated using the Khamis-Roche method to calculate percent of Predicted Adult Height (PAH), categorised as: pre-peak height velocity (PHV) (<88%), circa-PHV (88–93%), and post-PHV (>93%). Functional assessments included countermovement jump height, hip rotation, hamstring flexibility, and ankle mobility. Temporal associations with OSD onset were assessed using generalised linear mixed-effects models on a lagged dataset. Mediation analyses were conducted evaluating the temporal relationships between associated variables.

Results: OSD risk peaked at 88.3% PAH (p=0.02). A linear increase in risk was observed with growth rate for stature (OR = 1.16, p=0.006) and leg length (OR = 1.16, p=0.011). Hip external rotation was negatively associated with OSD risk (OR = 0.94, p=0.012). Exploratory trends were observed for hip internal rotation (p=0.069) and hamstring flexibility (p=0.088). Mediation analysis indicated no temporal relationship between growth rate and hip external rotation (p=0.464).

Conclusion: OSD risk increases linearly with growth rate and peaks during the Circa-PHV period. Reductions in specific physical functions may also be associated with elevated risk.

KEYWORDS: ADOLESCENCE, PUBERTY, INJURY PREVENTION, SOCCER, APOPHYSIS, BONE HEALTH

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INTRODUCTION

Football academies play a crucial role in developing young talent and preparing players for elite competition. To support this process, academies implement holistic development pathways designed to optimise performance while mitigating injury risk (1, 2). Despite these efforts, the high physical and psychological demands of academy football can contribute to both acute and gradual-onset injuries. Such injuries often result in significant time loss, disrupting training continuity and limiting long-term development and progression to senior football (3). Given the substantial financial, performance, and developmental costs

associated with injury (4, 5), effective monitoring and injury prevention strategies are critical to ensuring athlete success. (6, 7). Growth and maturation are key markers of a developmental period associated with injury risk in players, as youth athletes undergo rapid and individualised structural and neuromuscular changes (8). Due to their immature musculoskeletal systems, young athletes are particularly susceptible to growth-related injuries such as physeal fractures and apophysitis. These injuries are common in high-level youth football and have been reported to account for up to 21% of all injuries sustained between Under-11 (U11) and Under-19 (U19) age groups (9, 10). The burden of such injuries is highest around the period of Peak Height Velocity (PHV), with nearly half of all time-loss injuries occurring between 85% and 96% of predicted adult height (10).

One of the most frequently observed growth-related conditions in this context is Osgood-Schlatter's Disease (OSD), characterised by repetitive loading of the patellar tendon on the developing tibial tuberosity (11, 12). OSD is often considered self-limiting (13), with symptoms typically coinciding with the appearance and ossification of the tibial tuberosity (15), presenting between the ages of 8–12 in girls and 12–15 in boys (16, 17), and potentially persisting for more than two years. In severe cases, athletes may self-limit their activity or withdraw from sport altogether (18, 19). While activity modification and graduated return-to-play strategies have shown success in reducing symptoms (20), the common recommendations to reduce training load, or temporarily withdraw from sporting activity over these prolonged periods, can compromise physical development and may increase the risk of symptom recurrence upon reintroduction to full training (21, 22). Furthermore, merging evidence suggests that OSD may lead to long-term impairments in strength, motor control, and sport participation (14).

Previous studies have identified associations between OSD and both rapid growth tempo (e.g., growth rates >7.2 cm/year) and critical maturation windows (e.g., 85–91% of predicted adult height) (10, 23, 24). Additionally, functional deficits, including reduced quadriceps and gastrocnemius flexibility (25-27), and altered hamstring-to-quadriceps strength ratios (28), have been proposed as potential risk factors. However, most research to date has been cross-sectional, limiting the ability to determine whether such characteristics are risk factors for, or consequences of, OSD.

To address the current limitations within both knowledge and literature pertaining to OSD, the present study adopts a nested case-control design, comparing male Premier League academy footballers with OSD symptoms to controls. The primary objective of this study is to identify functional, growth, and maturation-related risk factors associated with OSD onset. The findings from this study may inform evidence-based monitoring and prevention strategies to better support the health and performance of youth footballers during critical stages of development.

METHODS

Study design and setting

This study was designed as a nested case-control study based in an English Premier League academy. The study was approved by the ethics committee of the host institution (application reference number: 5725-6727) and prospectively registered on OSF prior to data analysis (ref: osf.io/whkyx).

Participants

A total of 254 male youth football players enrolled in a Premier League Academy between September 2019 and October 2024 assented to data collection, obtained written parental or guardian consent, and were considered for inclusion. Players represented seven age groups (U12 to U18), with longitudinal data spanning five years and one month.

To evaluate temporal changes in key outcomes, inclusion required a minimum of three consecutive data collection points (equivalent to approximately nine months of continuous monitoring). Players were excluded if they: did not meet the minimum monitoring threshold, lacked accessible parental height data necessary for Khamis-

Roche maturity status estimation, reported a history of Osgood-Schlatter disease (OSD) symptoms prior to baseline data collection, collected via online survey (details included in supplementary material), or had an unknown OSD status due to non-response or having left the academy before follow-up.

Osgood-Schlatter's Disease Diagnosis

Youth players at the club who reported OSD symptoms (pain around the tibial tuberosity) were assessed by a trained member of the medical team at the football club. Diagnosis was based on a comprehensive clinical assessment, consistent with established practice (29-31), details of which can be found within the supplementary material. Diagnosis was recorded using the club's online database (Smartabase, Fusion Sport, Brisbane, Australia). Once diagnosed, players underwent weekly monitoring assessments to evaluate pain and range of motion following standardised club protocols.

Growth and Maturation

Anthropometrical assessment of stature, sitting height, and mass was taken every 12 weeks following ISAK-recommended procedures (32) using calibrated equipment (Seca GmbH & Co. KG, Hamburg, Germany). Seated height was subtracted from the stature to estimate leg length. Growth rates were calculated for each player as the change in height for stature or leg length, divided by the days between assessments, multiplied by 365, giving a rate in cm per year. Stature, mass, chronological age, and mid-parent stature were used to predict the adult height of each player via the Khamis-Roche method (33). At each time-point, height expressed as a percentage of predicted adult height (%PAH) was calculated as an index of somatic maturity (34).

Maturity status was categorised into the following bandings for description; pre-PHV (<88% PAH), circa-PHV (88% to 93% PAH), and post-PHV (>93% PAH) which show a high concordance (35). Maturity timing was calculated for each measurement time-point by subtracting their current %PAH from the median age associated with that %PAH in the UK1990 reference data (36). They were then categorised into three groups for description: early (>0.5 years), on-time (-0.5 to +0.5 years), or late (<-0.5 years).

Physical Tests

Physical tests were collected in accordance with the club's testing and screening protocols. Strength data was collected by sports science and medical staff at the Premier League club every three months throughout the season, in line with Premier League Guidelines (5).

Functional variables included countermovement jump height, hip internal and external rotation, hamstring flexibility, and weight-bearing dorsiflexion range of motion. All assessments were conducted by trained medical or performance staff using standardised protocols at routine testing timepoints. Detailed assessment protocols are provided in the Supplementary Material. Countermovement jump height, used as a proxy for lower-body strength, was assessed using the OptoJump system (OptoJump, Microgate, Bolzano, Italy), with players performing three maximal efforts following a standardised warm-up. Flexibility measures were obtained using passive manual assessments using a universal goniometer following standard procedures (37): hip rotation in prone, hamstring flexibility in supine, and ankle dorsiflexion via a weight-bearing lunge test. Values were expressed as the maximum or last successful attempt for each measure. Change scores between timepoints were used for analyses.

Statistical Analysis

To evaluate the prospective effects of temporal changes in predictor variables on subsequent outcomes, we constructed a lagged dataset reflecting repeated measures collected at ~12-week intervals ('Test Windows'). For each participant, observations were chronologically ordered, and first-order change scores were computed for physical tests as the difference between each variable's value at time t and $t - 1$ (e.g., Test Window B − Test Window A). These change scores were then used as fixed-effect predictors of the outcome measured at time $t + 1$ (e.g., Test Window C). Analyses were conducted in R Statistical Software (v4.3.2; R Core Team 2021) using the '*lme4*', '*sjPlot*', '*mediator*' and '*performance*' packages. Generalized Linear Mixed Models (family = *binomial*) were used to model the relationship

between potential associated factors and the onset of OSD symptoms in a univariate fashion. Non-linear relationships for continuous variables were explored by including polynomial terms and were retained when the squared term was significant (38). Significant predictors were then included in multivariable models to visualise their combined effects via heatmaps, as per Johnson et al, (39). Growth rate variables were included alongside the strength and mobility predictors to control for the effect of maturation. Player ID was included as a random effect to account for clustering of observations. Odds ratios and 95% confidence intervals (CI) were calculated, with statistical significance determined using an alpha value of 0.05. For exploratory analyses involving the physical tests, an alpha value of 0.10 was used to reduce the risk of type II errors.

Post hoc analyses were performed to investigate whether changes in hip external rotation range of motion mediated the relationship between prior growth rate and the onset of Osgood-Schlatter disease, a causal mediation analysis was performed using the counterfactual framework within the 'mediator' package in R Statistical Software (v4.3.2; R Core Team 2021).

The mediation model was structured to ensure appropriate temporal ordering of variables. Specifically, the predictor was double-lagged growth rate (calculated as the change in height velocity between timepoints t-3 and t-2), the mediator was hip external rotation change (from t-2 to t-1), and the outcome was OSD onset at time t. This lag structure was used to reduce the risk of reverse causation and satisfy temporal assumptions.

RESULTS

A sample of 88 players met all the eligibility criteria, comprising 48 players who developed OSD during the monitoring period and 40 controls. A detailed flowchart outlining participant inclusion and exclusion is presented in Figure 1. The average chronological age at OSD onset was 13.38 years and the mean duration of symptoms was 15.5 months (range 0.7 to 32.2).

Growth & Maturation as a Risk Factor for OSD

Percentage of predicted adult height was significantly associated with the likelihood of OSD onset, with evidence of a non-linear relationship with peak risk occurring at 88.3% PAH (Figure 2). The linear model indicated an initial increase in odds of OSD onset (OR = 11.38, 95% CI: 1.48–87.77, p=0.02). However, the quadratic term was also significant (OR = 0.99, 95% CI: 0.98–1.00, p=0.02), suggesting a curvilinear effect.

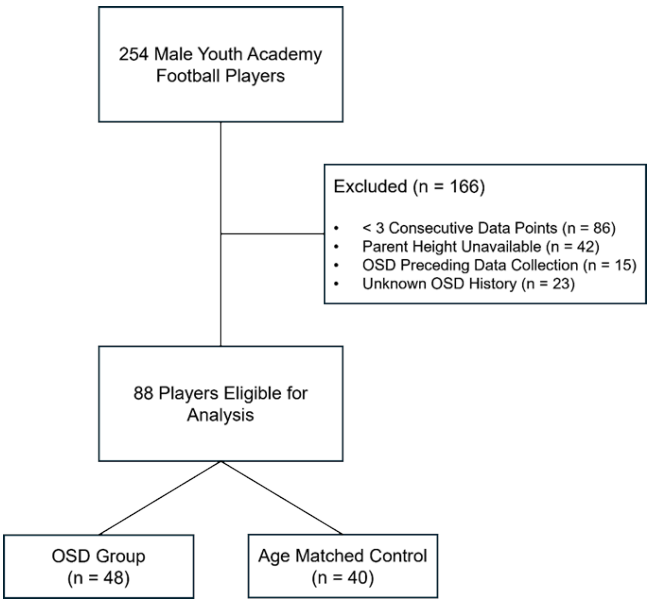

FIGURE 1. Flow chart of study participants.

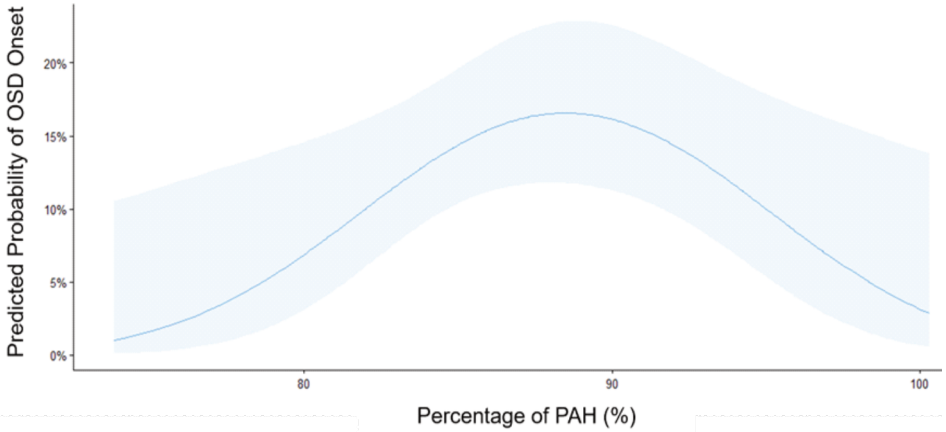

FIGURE 2. The estimated risk of OSD onset compared to lagged percentage of predicted adult height (%PAH). (The blue line represents the estimated risk, and the light blue shaded area represents the 95% confidence intervals).

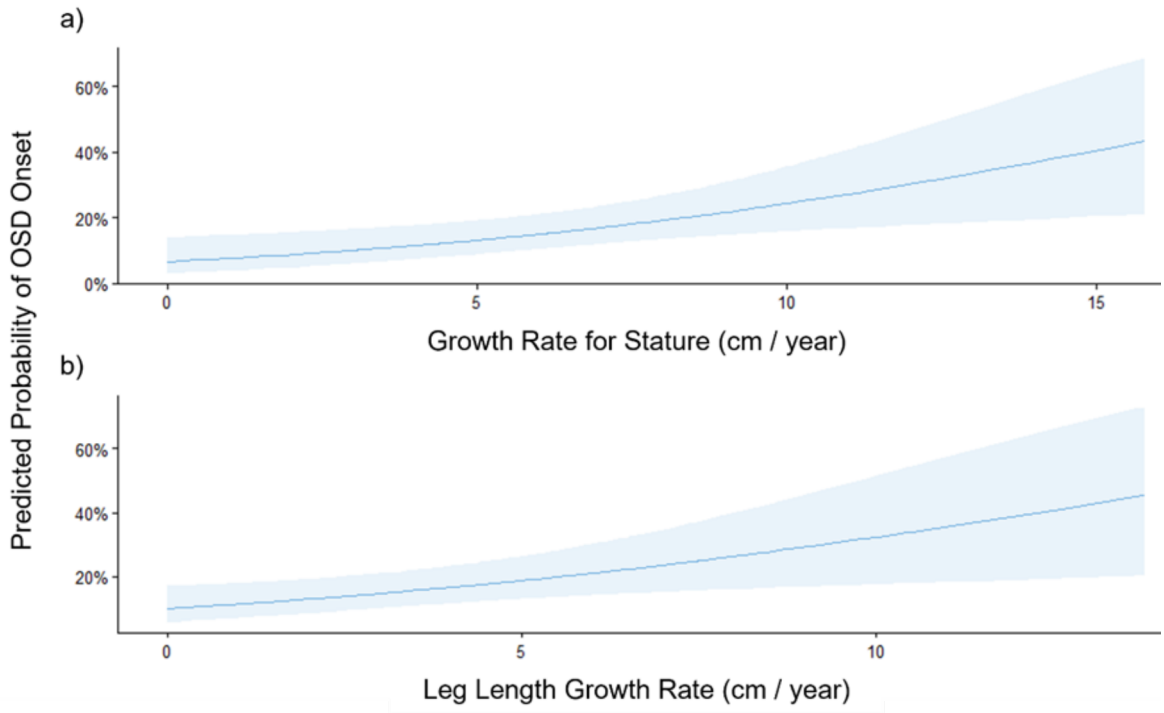

FIGURE 3. a) The estimated risk of OSD onset compared to lagged growth rate per year (cm / year). b) The estimated risk of OSD onset compared to lagged leg length growth rate per year (cm / year). (The blue line represents the estimated risk, and the light blue shaded area represents the 95% confidence intervals)

Higher growth rates for stature, and leg length, were significantly associated with increased odds of OSD onset. Specifically, for each cm/year increase in lagged growth rate for stature, the odds of developing OSD increased by 16% (OR = 1.16, 95% CI: 1.04-1.29, p=0.006) and 16% for leg length growth rate (OR = 1.16, 95% CI: 1.03–1.29, p=0.011), respectively (Figure 3). The mean growth rate for stature and leg length were 5.62 (± 3.47) cm/year and 3.1 (± 2.9) cm/year, respectively. The combined effects of growth rate for stature and percentage PAH, as well as leg length growth rate and percentage PAH, on the likelihood of OSD onset are displayed as heat maps in Figure 4.

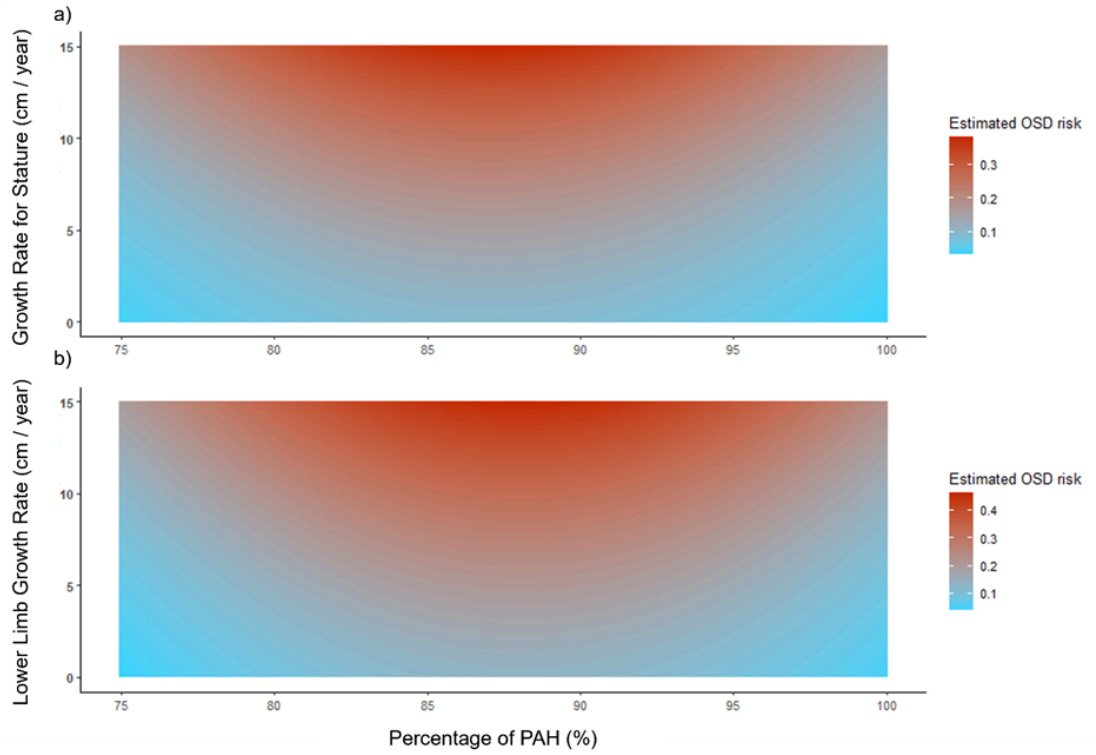

FIGURE 4. a) The combined effects of growth rate (cm/year) and percentage of PAH (%) on the onset of OSD. b) The combined effects of lower limb growth rate (cm/year) and percentage of PAH (%) on the onset of OSD.

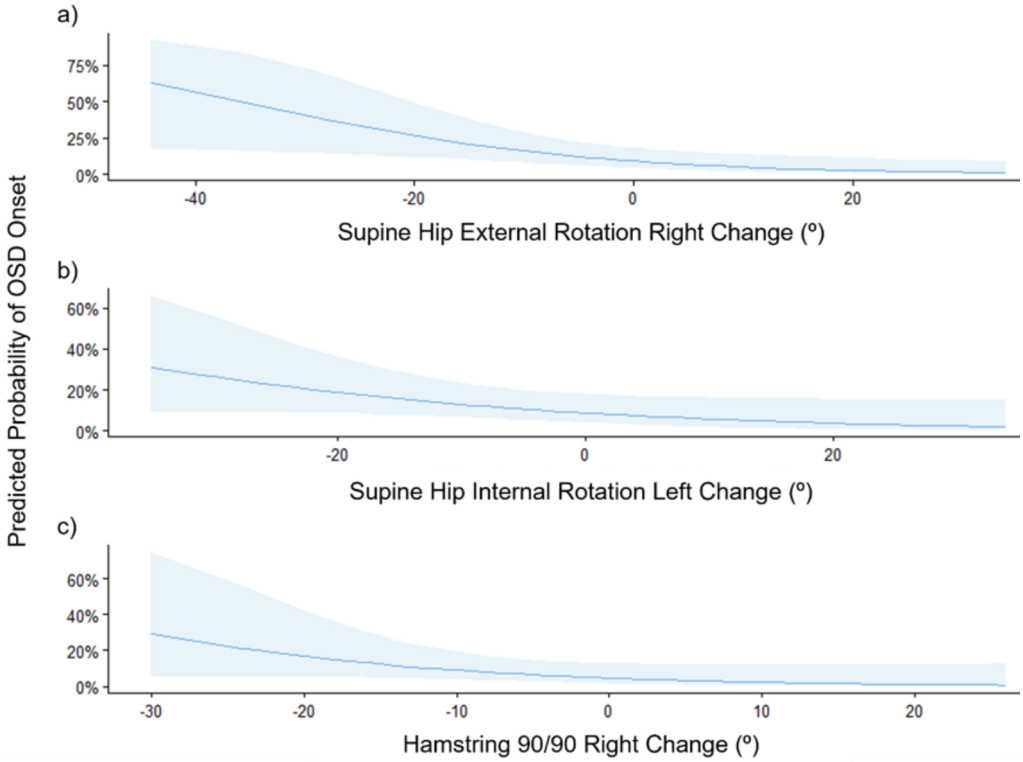

FIGURE 5. a) The estimated risk of OSD onset compared to lagged change in supine hip external rotation of the right limb (°). b) The estimated risk of OSD onset compared to lagged change in supine hip internal rotation of the left limb (°). c) The estimated risk of OSD onset compared to lagged change in hamstring 90/90 flexibility of the right limb (°). (The blue line represents the estimated risk, and the light blue shaded area represents the 95% confidence intervals)

Functional Risk Factors for OSD

The change in supine hip external rotation on the right limb demonstrated a significant negative linear relationship with the onset of OSD (OR = 0.94, 95% CI: 0.89–0.99, p=0.012). Change in supine hip internal rotation of the left limb and change in hamstring 90/90 flexibility on the right limb, both displayed linear trends with the onset of OSD (OR = 0.96, 95% CI: 0.91–1.00, p=0.069, and OR = 0.93, 95% CI: 0.86–1.01, p=0.088, respectively) (Figure 5). No other functional variables were significantly associated with OSD onset. A summary of findings can be found in Table 1.

TABLE 1. Results of generalised linear mixed modelling for variables in a lagged dataset.

	Odds ratio	95% CI	*P*-value
Growth & Maturation			
Growth Rate for Stature	1.16	1.04–1.29	0.006*
Growth Rate for Leg Length	1.16	1.03–1.29	0.011*
Physical Tests			
Countermovement Jump Height Δ	1.06	0.97–1.17	0.192
Supine Hip External Rotation Left Δ	0.96	0.90–1.02	0.208
Supine Hip External Rotation Right Δ	0.94	0.89–0.99	0.012*
Supine Hip Internal Rotation Left Δ	0.96	0.91–1.00	0.069†
Supine Hip Internal Rotation Right Δ	0.97	0.91–1.03	0.323
Prone Hip Internal Rotation Left Δ	0.99	0.95–1.04	0.822
Prone Hip Internal Rotation Right Δ	0.96	0.90–1.03	0.218
Hamstring 90/90 Left Δ	0.99	0.93–1.06	0.819
Hamstring 90/90 Right Δ	0.93	0.86–1.01	0.088†
WBDF Left Δ	0.98	0.67–1.44	0.914
WBDF Right Δ	0.93	0.64–1.34	0.685

*Δ = change score, WBDF = weight-bearing dorsiflexion, *=p< 0.05, †=p<0.10.*

When growth rate and supine hip external rotation on the right limb were modelled together, hip external rotation remained statistically significant (p=0.013), but growth rate did not (p=0.051). No other functional variables remained significant when modelled alongside growth rate for stature or leg length.

Post-hoc analysis investigating the mediating effects of hip external rotation on growth rate to OSD pathway revealed no significant indirect (ACME = 0.0003, p=0.846) or direct effects (ADE = −0.0114, p=0.436). The total effect of growth rate on OSD was also non-significant (p=0.464).

DISCUSSION

This study aimed to identify physical and growth and maturation-related factors associated with OSD onset in Premier League male academy football players. From retrospective analysis of data collected over five years, we found that periods of rapid growth during key maturational stages, as well as specific functional deficits in hip and hamstring flexibility, appear to be associated with a greater risk of developing OSD.

The median duration of OSD symptoms was 15.5 months (IQR: 7.9–23.3), aligning with previous research in youth football populations (25, 30). Although a shorter mean duration might have been anticipated given the continuous medical supervision provided within the academy setting, this effect may have been counterbalanced by the higher training volume and intensity relative to the populations studied previously. Furthermore, it is important to acknowledge that earlier studies relied on self-reported symptoms and duration, which may be less precise than the medical diagnosis and monitoring applied in the present study (25, 30).

Growth & Maturation as a Risk Factor for OSD

Risk of developing OSD appears to peak at a median of 88.3% PAH (IQR: 85.5% - 89.5%), aligning with previous research (10). The rapid increase in rate of growth during adolescence occurs between 88% and 93% of predicted adult height (35), and has previously been linked to increased risk of injury (24, 39). While chronological age is commonly used to denote developmental stage in team sports settings, it does not account for individual variability in biological maturation and is therefore limited when reporting a development associated condition. Therefore, our study focused on indicators of biological maturation rather than relying on chronological age.

Growth rates for stature and leg length were also associated with the onset of OSD. Following a linear trend, a 1 cm/year increase in growth rate resulted in a 16% increase in risk. For example, a player growing at 10.6 cm/year would be twice as likely to develop OSD compared to a player on the same age and maturation status growing at 5.6 cm/year. These findings are consistent with biomechanical theories suggesting that accelerated long bone growth increases both the mass and length of bones, significantly raising joint moments of inertia (40).

Our findings reinforce growth rate as a factor associated with overuse injuries which has been highlighted in previous studies (41, 42). It is important, however, to acknowledge inconsistencies in the literature regarding growth rate as one study, for example, failed to detect this relationship (43). This was potentially due to a reliance on the Mirwald maturity offset method to estimate PHV timing (44), a method that has shown to misclassify the timing of the growth spurt in approximately half of young athletes (35). The use of more precise methods combined with repeated anthropometric assessments strengthens the validity of our findings.

This period of rapid growth also corresponds with the maturation process of the tibial tuberosity which begins around 11 years of age and develops until approximately 17 years of age in males (15, 45). Studies have shown that OSD more commonly appears during the early apophyseal stage of maturation, where a visible ossification centre appears (46). It is thought that throughout this process, the open physis is in a relatively weaker and more malleable state, and more sensitive to repetitive and forceful traction (47).

This current study adds to the body of evidence associating rapid growth and maturation timing with an increased risk of

developing OSD onset in Premier League academy footballers. Although the causal mechanisms remain unknown, it appears likely that rapid growth and the subsequent elevated force requirements contribute to increased stress placed on a vulnerable apophyseal site.

Functional Risk Factors of OSD

Alongside growth and maturation-related markers, we explored whether specific functional deficits contributed to OSD onset. A larger alpha value of 0.10 identified significant associations between OSD risk and reduced supine hip external rotation on the right side, supine hip internal rotation on the left side, and hamstring range of motion on the right.

Reduction in range of motion of the lower extremities likely corresponds with the period encompassing peak height velocity (46). This increase in long bone growth and the associated non-uniform development of soft tissue can lead to increased tension within the musculotendinous unit which would likely explain this occurrence of reduced range of motion in the lower extremities (31, 48, 49). Previous research has demonstrated asymmetrical hip kinematics during the soccer kicking motion, whereby the kicking limb typically exhibits greater external rotation, while the stance limb functions primarily to stabilise the hip and displays greater internal rotation (50, 51). Although limb dominance was not recorded in the present study, previous work indicates that approximately 82–85% of youth soccer players are right-limb dominant (52, 53). Accordingly, it is plausible that reduced external rotation on the right (kicking) side and reduced internal rotation on the left (stance) side may interfere with optimal kicking kinematics, causing an observed shifting of the centre of mass during kicking motions (26). Such alterations have been associated with increased rotational and shear loading at the knee joint (54, 55), potentially explaining the observed side-specific differences in injury risk.

Football-specific actions such as cutting, decelerating, and kicking, particularly with the non-dominant leg, create high levels of angular impulse at the knee joint. This is further exacerbated as a consequence of the growth spurt, with some studies estimating a 30% increase in required force to execute the same movement (56). Furthermore, single-leg loading tasks are also associated with increased pain and exacerbated OSD symptoms, as reported in related work (57). Single-leg landings, squats, and jumps place considerable load through the patellar tendon, which may explain why such movements, commonly performed in football-specific tasks, frequently exacerbate symptoms. The shift in centre of mass during kicking observed in symptomatic players, could be indicative of the reduced range of motion at the hip. This likely increases quadriceps activation and strain on the tibial tuberosity of the stance leg further, reinforcing this as a potential mechanism for OSD.

Although not assessed during this study, reduced quadricep flexibility has been found to be a significant factor in the onset of OSD (25, 26, 58, 59). The asynchronous elongation of the femur during growth, combined with relative asynchronous development of the rectus femoris (31), likely contributes to increased compressive forces on the tibial tuberosity. This, combined with altered kicking strategies, resulting from reduced hip range of motion, could explain why this appears as a risk factor for OSD.

In post hoc analysis, hip external rotation remained significantly associated with OSD after accounting for growth rate. Conversely growth rate failed to demonstrate a significant independent association with OSD when controlling for hip external rotation Further mediation analysis did not support a causal pathway through which growth rate affected OSD via hip flexibility, with only 2% of the total effect explained by this route. Whilst these findings suggest that growth rate and hip external rotation may act as independent risk factors for OSD, the causal mechanisms remain unknown; however, we cannot rule out the influence of biomechanical or growth-related mechanisms on the relationships between these variables and OSD. it must be interpreted with caution as the post-hoc analysis required 4 consecutive time points which impacted the power of the analysis. Further longitudinal research employing causal inference modelling is warranted to clarify the temporal and mechanistic contributions to OSD.

While prior studies have reported mixed results regarding the role of reduced joint range of motion in OSD (16, 28, 60), our longitudinal approach enables temporal comparison, providing clearer insights into the timing of functional changes associated with OSD onset. Many earlier studies relied on cross-sectional designs or single-point assessments (16, 58, 61), limiting their ability to discern whether reduced flexibility preceded, or was consequent to, injury onset. Our within-subject longitudinal approach addresses this limitation and provides stronger evidence that reduced flexibility may indeed precede OSD development.

CONCLUSION

This study offers further insights into the potential antecedents of Osgood-Schlatter's Disease in Premier League academy footballers. By examining longitudinal changes and their temporal relationship to OSD onset, it enhances the understanding of how adolescent athletes respond to growth-related factors and functional adaptations. These findings highlight the importance of regular growth monitoring and targeted screening of functional deficits during critical periods of maturation to identify players at an increased risk of OSD. Early detection can lead to the implementation of appropriate injury mitigation strategies, focusing on flexibility, neuromuscular control, and load management to reduce stress placed on the tibial tuberosity, to create a shift towards proactive, individualised management.

This study reflects data from a single Premier League academy, limiting generalisability to other settings. Variations in testing protocols, player availability, and data storage systems over the five-year period led to some inconsistencies and missing data. To enable temporal analysis, strict inclusion criteria were applied, resulting in a smaller sample size and reduced statistical power, requiring an exploratory alpha level of 0.10 to minimise type II errors.

The available variables were constrained by the academy's screening protocols, with no direct measures of quadriceps strength or flexibility included. Countermovement jump height was used as a proxy for lower-body strength, though this measure is influenced by multiple factors beyond muscular force production. Additionally, while ankle dorsiflexion was assessed, the absence of detailed biomechanical or kinematic analysis in this study limits interpretations of joint mechanics and their perceived role in OSD onset.

Further longitudinal research studies should continue investigating associated factors of OSD across multiple academy environments and utilise causal inference models to determine the mechanisms driving OSD onset.

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ETHICAL APPROVAL: The study was approved by the ethics committee of the host institution (ref: 5725-6727)

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