Exploring Growth, Maturity, and Age as Injury Risk Factors in High-Level Youth Football

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ABSTRACT
Rapid somatic growth and biological maturity status may affect injury patterns in youth football, yet firm conclusions cannot be drawn from the existing research. We aimed to explore growth velocity, maturity, and age as injury risk factors in 95 academy players (11.9–15.0 years), using anthropometric (height and body mass), maturity (skeletal age), injury, and football exposure data collected prospectively over three seasons (2016/17–2018/19). We compared the relative quality of mixed-effects logistic regression models with growth velocity for 223 growth intervals (average 113 days) included as fixed effects and adjusted for age (chronological or skeletal) plus load (hours/week). Associations were considered practically relevant based on the confidence interval for odds ratios, using thresholds of 0.90 and 1.11 to define small beneficial and harmful effects, respectively. We observed harmful effects of older age on overall (OR: 2.61, 95% CI: 1.15–5.91) and sudden onset (1.98, 1.17–3.37) injury risk. Significant associations (p < 0.05) were observed for higher body mass change and greater maturity on sudden onset injuries, and for higher hours/week on gradual onset, bone tissue, and physis injuries. Future studies should include larger samples, monitoring athletes from preadolescence through maturation, to enable within-subject analyses and better understand the relationship between growth, maturation, and injuries.
Introduction

Elite football (soccer) academies guide players through structured and intensive training programs to optimize long-term development. Injuries impact these opportunities and identification of risk factors is an important step towards reducing injury occurrence and severity by informing targeted injury risk reduction strategies [1]. In youth football, emerging evidence suggests an association between growth, maturation, and injury risk [2, 3]. However, further research is needed to better understand these relationships and how the impact of injuries may be reduced during periods of rapid changes to an athlete’s body.

Growth represents an increase in the size of the body as a whole or of a specific body part, assessed using anthropometric measures [4]. An accelerated period of somatic growth is observed during the adolescent years, with peak height velocity (PHV) and peak weight velocity (PWV) for an average boy occurring around the age of 13 to 14 years and 14 to 15 years, respectively [4]. There is, however, a wide range in both timing (age at PHV from 12 to 17 years) and intensity (PHV from 5 to 12 cm/year) [5, 6]. Studies in high-level football have indicated that phases characterized by rapid growth are associated with greater overall, overuse, acute, and non-contact injury risk [7–14], and that players with faster PHV have a greater overall and growth-related injury burden compared to players with average or slower peak growth rates [15]. Still, it is difficult to provide clear recommendations as the associations are inconsistent and methodological differences make direct comparisons or pooling of results impossible.

Maturation is defined by Malina et al. [4] as the process of becoming mature or progressing towards a mature (adult) state (e.g., a fully ossified skeleton, adult height, or a functional reproductive system). Timing and tempo vary greatly between individuals (onset of puberty from 9 to 14 years in boys) [4], and the maturity status (where an individual is in the process at a given point) of two players who train and compete in the same age group can therefore differ substantially [4]. Only a few studies have related skeletal maturity – considered the single best marker of biological maturity [16] – to injury risk in high-level youth football [17–20]. While suggesting that maturity plays a role in the occurrence of certain injury types (e.g., apophyseal or osteochondral injuries, muscle injuries, joint/ligament injuries) [18–20], results are not consistent. Observed age-related injury patterns do, however, indicate that older players are at greater risk and that different pathology types are more prominent in younger, compared to older, age groups [21–23].

Since firm conclusions cannot be drawn from the current pool of literature, the aim of this study was to explore growth velocity, skeletal maturity, and chronological age as injury risk factors in male academy players. More specifically, our main research question was whether changes in height or body mass between assessments at the start, middle, and end of a football season were related to the occurrence of specific injury types when also taking age (chronological or skeletal), growth × age/maturity interaction effects, and football exposure into consideration. As an exploratory study, no a priori hypothesis was stated.

Materials and Methods

Study design and participants

We used injury, individual training and match exposure, anthropometric and maturity data collected prospectively over three seasons (2016/17 through 2018/19) in the U13, U14, and U15 age groups at one elite national football academy in Qatar (2016/17: 64 players in the program, 2017/18: 77 players, 2018/19: 91 players). Participants were boys aged 11 to 15 years and full-time players typically participated in eight sessions during the school week, in addition to local club games on weekends. Part-time players participated in five sessions in addition to weekend club games. Written informed consent to use routinely collected monitoring data for research purposes was obtained from the players’ guardians and ethics approval was granted from the Anti-Doping Lab Qatar Institutional Review Board (IRB Application #2014000012).

Recording of injuries and football exposure

Training and match injuries were recorded by the designated team physiotherapist (i.e., one physiotherapist per team) who was present at all team sessions, supervised by two researchers. Recording procedures followed the recommendations from Fuller et al. [24], including only time-loss injuries (to reduce bias associated with using several clinical recorders over multiple seasons [25]), i.e., any physical complaint leading to the medical staff partially or fully restricting participation in future football activities. Diagnoses were confirmed by a sports medicine physician (employed full-time at the academy) in collaboration with the treating physiotherapist and reported based on the Sports Medicine Diagnostic Coding System (SMDCS) [26], alongside details about the date of injury and mechanism. No inter-rater reliability data is available regarding the diagnosis or classification of injuries during the observation period. Each team’s designated sports scientist recorded individual training and match exposure.

Following the completion of the data collection, a researcher converted injury diagnoses to the updated SMDCS categories for tissue and pathology types [27, 28], and retrospectively allocated onset based on the reported mechanism and diagnosis following the consensus recommended definitions by Bah et al. ("sudden": resulting from a specific identifiable event, or "gradual": lack of definable sudden, precipitating event) [28]. Only index injuries were considered for this study while recurrent injuries were excluded; these were defined as a time-loss injury to the same location of the same type as a previous injury recorded during the observation period [28].

Anthropometric and maturity assessments

Measures of standing height and body mass were obtained at the start, middle, and end of each academy season by trained sports scientists following the recommendations outlined by Stewart et al. [29]. Measures were taken in the morning, prior to any activities to minimize diurnal variations. Standing height was measured to the nearest 0.1 cm applying the stretch stature method using a wall-mounted stadiometer (Holtain Ltd, Crymych, UK) and body mass was measured to the nearest 0.1 kg using digital scales (Adam Equipment, Milton Keynes, UK). Previously published test-retest data in a subsample of 17 academy players revealed a standard error of measurement (SEM) of 0.34 cm (95% confidence interval
(CI): 0.25 to 0.52 cm) for standing height [30]. This corresponds to a minimal detectable change (MDC) of approximately 1 cm.

Skeletal maturity was assessed at the beginning of the season using x-ray images of the player’s left hand/wrist complex taken at Aspetar Orthopaedic and Sports Medicine Hospital. Skeletal age was determined using the Fels method [31] by one trained assessor. Intra-rater reliability for this method has previously been reported (intraclass correlation coefficient (ICC): 0.998, 95 % CI: 0.996 to 0.999) [17].

**Inclusion and exclusion of growth intervals**

It has been suggested that researchers examine growth over shorter periods of time to better account for non-linear growth patterns related to saltatory (episodic) growth [32]. To minimize the impact of measurement error on estimations and allow for detection of meaningful changes [32, 33], we calculated growth velocity per academy semester, defined as the two intervals from season start (August/September) to mid-season (January) and from mid-season to season end (May/June). The absolute change (cm or kg) was divided by the number of days between measurements and converted to expressions equivalent to cm/year and kg/year, respectively [12, 13]. For a growth interval to be included in the final analyses, a skeletal maturity assessment had to be available for the given player and season (assessed maximum 91 days within the start of the season).

**Statistical analyses**

Descriptive statistics are presented as means with standard deviation (SD). Four separate mixed-effects logistic regression models (xtmelogit command) estimated associations for the effects of changes in height and body mass on the occurrence of overall, gradual onset, sudden onset, bone tissue and physis injury. Growth velocity for height and body mass were specified as distinct growth-related predictor variables (fixed effects). Models were adjusted for chronological or skeletal age, and growth × age/maturity interaction, with player specified as a random effect plus a random intercept. The average weekly load (hours of training/match exposure per week) during the growth interval (or until the event if an injury occurred) was added as a covariate.

The Akaike Information Criterion (AIC) assessed the relative quality of each mixed-effects logistic regression model in the set of candidate models. The Akaike difference (ΔAIC) from the estimated best model (i.e., the model with the lowest AIC value; ΔAIC = 0) was evaluated according to the following scale: 0–2, essentially equivalent; >2–7, plausible alternative; >7–14, weak support; >14, no empirical support [34]. Akaike weights (w) provided a scaled interpretation about the relative quality of each competing model as the probability that a given model is the best in the set of four candidate models per endpoint. Thresholds for the adjusted odds ratios (OR) of 0.9, 0.7, 0.5, 0.3, and 0.1 and their reciprocals 1.11, 1.43, 2.0, 3.3, and 10 defined small, moderate, large, very large, and extremely large beneficial and harmful effects, respectively [35]. In the absence of an established anchor defining practically relevant associations between growth velocity and injury occurrence, we considered OR > 0.90 or OR = 1.11 to define substantially beneficial and substantially harmful effects, respectively [35]. Associations were declared practically relevant based on the location of the confidence interval for the estimated ORs to these thresholds. Outcome statistics are reported as point estimates and 95 % CI. Statistical analyses were performed using Stata (StataBE v17.0; StataCorp, College Station, TX, USA).

**Results**

**Inclusion of players and growth intervals**

The inclusion of player-seasons and growth intervals are shown in Fig. 1. with an overview of exclusions due to incomplete assess-
Table 1  Overview of the 161 index injuries sustained within the growth periods included in the analyses, structured by location, onset, and pathology type.

<table>
<thead>
<tr>
<th>Location</th>
<th>Pathology type</th>
<th>Injuries (count)</th>
<th>Incidence (Inj. per 1000 h, 95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head and neck</td>
<td>Sudden onset</td>
<td>6</td>
<td>0.28 (0.10 to 0.60)</td>
</tr>
<tr>
<td></td>
<td>Brain/spinal cord</td>
<td>5</td>
<td>0.23 (0.07 to 0.54)</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>1</td>
<td>0.05 (0.00 to 0.26)</td>
</tr>
<tr>
<td>Upper limb</td>
<td>Sudden onset</td>
<td>18</td>
<td>0.83 (0.49 to 1.31)</td>
</tr>
<tr>
<td></td>
<td>Fracture</td>
<td>13</td>
<td>0.60 (0.32 to 1.02)</td>
</tr>
<tr>
<td></td>
<td>Contusion (superficial)</td>
<td>4</td>
<td>0.18 (0.05 to 0.47)</td>
</tr>
<tr>
<td></td>
<td>Joint sprain</td>
<td>1</td>
<td>0.05 (0.00 to 0.26)</td>
</tr>
<tr>
<td>Trunk</td>
<td>Sudden onset</td>
<td>7</td>
<td>0.32 (0.13 to 0.66)</td>
</tr>
<tr>
<td></td>
<td>Contusion (superficial)</td>
<td>2</td>
<td>0.09 (0.01 to 0.33)</td>
</tr>
<tr>
<td></td>
<td>Fracture</td>
<td>1</td>
<td>0.05 (0.00 to 0.26)</td>
</tr>
<tr>
<td></td>
<td>Non-specific</td>
<td>1</td>
<td>0.05 (0.00 to 0.26)</td>
</tr>
<tr>
<td></td>
<td>Gradual onset</td>
<td>3</td>
<td>0.14 (0.03 to 0.40)</td>
</tr>
<tr>
<td></td>
<td>Bone stress injury</td>
<td>2</td>
<td>0.09 (0.01 to 0.33)</td>
</tr>
<tr>
<td></td>
<td>Non-specific</td>
<td>1</td>
<td>0.05 (0.00 to 0.26)</td>
</tr>
<tr>
<td>Hip/groin</td>
<td>Sudden onset</td>
<td>26</td>
<td>1.20 (0.78 to 1.75)</td>
</tr>
<tr>
<td></td>
<td>Contusion (superficial)</td>
<td>4</td>
<td>0.18 (0.05 to 0.47)</td>
</tr>
<tr>
<td></td>
<td>Muscle injury</td>
<td>1</td>
<td>0.05 (0.00 to 0.26)</td>
</tr>
<tr>
<td></td>
<td>Non-specific</td>
<td>1</td>
<td>0.05 (0.00 to 0.26)</td>
</tr>
<tr>
<td></td>
<td>Gradual onset</td>
<td>22</td>
<td>1.01 (0.64 to 1.53)</td>
</tr>
<tr>
<td></td>
<td>Physis injury</td>
<td>20</td>
<td>0.92 (0.56 to 1.42)</td>
</tr>
<tr>
<td></td>
<td>Bone stress injury</td>
<td>1</td>
<td>0.05 (0.00 to 0.26)</td>
</tr>
<tr>
<td></td>
<td>Bursitis</td>
<td>1</td>
<td>0.05 (0.00 to 0.26)</td>
</tr>
<tr>
<td>Thigh</td>
<td>Sudden onset</td>
<td>35</td>
<td>1.61 (1.12 to 2.24)</td>
</tr>
<tr>
<td></td>
<td>Muscle injury</td>
<td>20</td>
<td>0.92 (0.56 to 1.42)</td>
</tr>
<tr>
<td></td>
<td>Muscle contusion</td>
<td>5</td>
<td>0.23 (0.07 to 0.54)</td>
</tr>
<tr>
<td></td>
<td>Non-specific</td>
<td>5</td>
<td>0.23 (0.07 to 0.54)</td>
</tr>
<tr>
<td></td>
<td>Cartilage</td>
<td>1</td>
<td>0.05 (0.00 to 0.26)</td>
</tr>
<tr>
<td></td>
<td>Gradual onset</td>
<td>4</td>
<td>0.18 (0.05 to 0.47)</td>
</tr>
<tr>
<td></td>
<td>Physis injury</td>
<td>3</td>
<td>0.14 (0.03 to 0.40)</td>
</tr>
<tr>
<td></td>
<td>Non-specific</td>
<td>1</td>
<td>0.05 (0.00 to 0.26)</td>
</tr>
<tr>
<td>Knee</td>
<td>Sudden onset</td>
<td>19</td>
<td>0.88 (0.53 to 1.37)</td>
</tr>
<tr>
<td></td>
<td>Contusion (superficial)</td>
<td>7</td>
<td>0.32 (0.13 to 0.66)</td>
</tr>
<tr>
<td></td>
<td>Joint sprain</td>
<td>2</td>
<td>0.09 (0.01 to 0.33)</td>
</tr>
<tr>
<td></td>
<td>Fracture</td>
<td>1</td>
<td>0.05 (0.00 to 0.26)</td>
</tr>
<tr>
<td></td>
<td>Non-specific</td>
<td>1</td>
<td>0.05 (0.00 to 0.26)</td>
</tr>
<tr>
<td></td>
<td>Gradual onset</td>
<td>8</td>
<td>0.37 (0.16 to 0.73)</td>
</tr>
<tr>
<td></td>
<td>Physis injury</td>
<td>5</td>
<td>0.23 (0.07 to 0.54)</td>
</tr>
<tr>
<td></td>
<td>Synovitis/capsulitis</td>
<td>2</td>
<td>0.09 (0.01 to 0.33)</td>
</tr>
<tr>
<td></td>
<td>Cartilage</td>
<td>1</td>
<td>0.05 (0.00 to 0.26)</td>
</tr>
<tr>
<td>Lower leg</td>
<td>Sudden onset</td>
<td>12</td>
<td>0.55 (0.29 to 0.97)</td>
</tr>
<tr>
<td></td>
<td>Muscle injury</td>
<td>11</td>
<td>0.51 (0.25 to 0.91)</td>
</tr>
<tr>
<td></td>
<td>Muscle injury</td>
<td>5</td>
<td>0.23 (0.07 to 0.54)</td>
</tr>
</tbody>
</table>

Table 1 Continued.

<table>
<thead>
<tr>
<th>Location</th>
<th>Pathology type</th>
<th>Injuries (count)</th>
<th>Incidence (Inj. per 1000 h, 95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle</td>
<td>Sudden onset</td>
<td>25</td>
<td>1.15 (0.75 to 1.70)</td>
</tr>
<tr>
<td></td>
<td>Gradual onset</td>
<td>5</td>
<td>0.23 (0.07 to 0.54)</td>
</tr>
<tr>
<td></td>
<td>Synovitis / capsulitis</td>
<td>5</td>
<td>0.23 (0.07 to 0.54)</td>
</tr>
<tr>
<td>Foot</td>
<td>Sudden onset</td>
<td>13</td>
<td>0.60 (0.32 to 1.02)</td>
</tr>
<tr>
<td></td>
<td>Contusion (superficial)</td>
<td>9</td>
<td>0.41 (0.19 to 0.79)</td>
</tr>
<tr>
<td></td>
<td>Fracture</td>
<td>2</td>
<td>0.09 (0.01 to 0.33)</td>
</tr>
<tr>
<td></td>
<td>Joint sprain</td>
<td>2</td>
<td>0.09 (0.01 to 0.33)</td>
</tr>
<tr>
<td></td>
<td>Gradual onset</td>
<td>4</td>
<td>0.18 (0.05 to 0.47)</td>
</tr>
<tr>
<td></td>
<td>Physis injury</td>
<td>2</td>
<td>0.09 (0.01 to 0.33)</td>
</tr>
<tr>
<td></td>
<td>Non-specific</td>
<td>2</td>
<td>0.09 (0.01 to 0.33)</td>
</tr>
</tbody>
</table>

The mean age at the start of a growth interval was 13.5 years (SD 0.8; range 11.9 to 15.0). Considering each player-season only once (a player could have two growth intervals per season but only one maturity assessment), the mean skeletal age at the start of the season was 14.4 years (SD 1.6) with skeletal ages ranging from 10.7 to 14.9 years in the U13 age group, 11.8 to 18.0 in U14, and 13.7 to 17.8 in U15. On average, players were 1.0 year (1.1; –1.5 to 4.7) advanced in skeletal age relative to chronological age. One player was skeletally mature (skeletal age 18 years), while 62 (45 %) could be considered early maturing (skeletal age minimum one year in advance of chronological age), 70 (51 %) as on time (skeletal age within one year) and four (3 %) as late maturing (skeletal age minimum one year delayed) [36]. The mean semester growth velocity...
was 6.3 cm/year (3.5; 0.0 to 17.8) for height and 5.3 kg/year (5.5; –14.6 to 19.3) for body mass.

Relative model quality

The relative model quality of the four model combinations within the five injury categories is presented in Table 2. Growth velocity for body mass combined with skeletal age best explained the overall and gradual onset injury risk, while change in height combined with skeletal age best explained the risk of sudden onset, bone tissue, and physis injuries. Other model combinations were, however, considered equivalent or plausible alternatives.

Effects of growth velocity, age, and skeletal maturity

Table 3 gives a complete overview of all model outcomes. Effects for load and growth × age/maturity interaction were not practically relevant for any injury categories. Practically relevant harmful effects of older age were observed for overall and sudden onset injury risk, while change in height combined with skeletal age best explained the risk of sudden onset injury risk. Significant associations were also found for greater change in body mass and more advanced maturity on sudden onset injury risk, and for greater football load on gradual onset, bone tissue, and physis injuries; however, these were not considered practically relevant based on our pre-defined thresholds. No significant growth × age/maturity interaction effects were seen.

Discussion

This study explored growth velocity and age (skeletal and chronological) as injury risk factors, accounting for growth × age/maturity interaction effects and individual training and match exposure. Based on prospective data from 95 unique players between 11 and 15 years over three seasons, we observed harmful effects of older age on overall and sudden onset injury risk. Significant associations were also found for greater change in body mass and more advanced maturity on sudden onset injury risk, and for greater football load on gradual onset, bone tissue, and physis injuries; however, these were not considered practically relevant based on our pre-defined thresholds. No significant growth × age/maturity interaction effects were seen.

Associations between growth velocity and injury risk remain unclear

A potential link between growth velocity and injury risk is typically attributed to underlying mechanisms such as tissues adapting at different rates, increased tension on apophyses, or decreased neuromuscular control [3, 37–39]. In support of such a link, associa-
Table 3  Odds ratios for the four model combinations within each of the five injury outcomes. Numbers in italics indicate significant associations (p<0.05), while asterisks indicate practically relevant findings based on our predefined thresholds (95% CI for OR < 0.9 or > 1.1).

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Model</th>
<th>Odds ratio (95% CI)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall</td>
<td>Δ Height (cm/year)</td>
<td>3.78 (0.79 to 18.03)</td>
<td>0.10</td>
</tr>
<tr>
<td></td>
<td>Chronological age</td>
<td>2.61 (1.15 to 5.91)</td>
<td>0.022*</td>
</tr>
<tr>
<td></td>
<td>Δ Height × chronological age</td>
<td>0.91 (0.81 to 1.02)</td>
<td>0.10</td>
</tr>
<tr>
<td></td>
<td>Hours per week</td>
<td>1.06 (0.94 to 1.21)</td>
<td>0.35</td>
</tr>
<tr>
<td></td>
<td>Δ Height (cm/year)</td>
<td>1.43 (0.59 to 3.43)</td>
<td>0.43</td>
</tr>
<tr>
<td></td>
<td>Skeletal age</td>
<td>1.40 (0.97 to 2.03)</td>
<td>0.07</td>
</tr>
<tr>
<td></td>
<td>Δ Height × skeletal age</td>
<td>0.98 (0.92 to 1.04)</td>
<td>0.44</td>
</tr>
<tr>
<td></td>
<td>Hours per week</td>
<td>1.06 (0.93 to 1.20)</td>
<td>0.38</td>
</tr>
<tr>
<td></td>
<td>Δ Body mass (kg/year)</td>
<td>1.82 (0.67 to 4.92)</td>
<td>0.24</td>
</tr>
<tr>
<td></td>
<td>Chronological age</td>
<td>1.65 (0.99 to 2.73)</td>
<td>0.05</td>
</tr>
<tr>
<td></td>
<td>Δ Body mass × chronological age</td>
<td>0.96 (0.89 to 1.03)</td>
<td>0.29</td>
</tr>
<tr>
<td></td>
<td>Hours per week</td>
<td>1.08 (0.95 to 1.22)</td>
<td>0.26</td>
</tr>
<tr>
<td></td>
<td>Δ Body mass (kg/year)</td>
<td>0.92 (0.54 to 1.58)</td>
<td>0.77</td>
</tr>
<tr>
<td></td>
<td>Skeletal age</td>
<td>1.21 (0.97 to 1.51)</td>
<td>0.09</td>
</tr>
<tr>
<td></td>
<td>Δ Body mass × skeletal age</td>
<td>1.01 (0.97 to 1.05)</td>
<td>0.59</td>
</tr>
<tr>
<td></td>
<td>Hours per week</td>
<td>1.06 (0.94 to 1.20)</td>
<td>0.36</td>
</tr>
<tr>
<td>Sudden onset</td>
<td>Δ Height (cm/year)</td>
<td>2.26 (0.53 to 9.58)</td>
<td>0.27</td>
</tr>
<tr>
<td></td>
<td>Chronological age</td>
<td>1.95 (0.93 to 4.10)</td>
<td>0.08</td>
</tr>
<tr>
<td></td>
<td>Δ Height × chronological age</td>
<td>0.94 (0.85 to 1.05)</td>
<td>0.26</td>
</tr>
<tr>
<td></td>
<td>Hours per week</td>
<td>1.06 (0.94 to 1.21)</td>
<td>0.33</td>
</tr>
<tr>
<td></td>
<td>Δ Height (cm/year)</td>
<td>1.36 (0.59 to 3.13)</td>
<td>0.47</td>
</tr>
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<td>Δ Body mass (kg/year)</td>
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Table 3 Continued.

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<td>Bone tissue</td>
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<td>Hours per week</td>
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<td>Δ Height (cm/year)</td>
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<td>Δ Body mass × chronological age</td>
<td>0.97 (0.90 to 1.06)</td>
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<td>Physis injury</td>
<td>Δ Height (cm/year)</td>
<td>1.76 (0.22 to 14.37)</td>
<td>0.60</td>
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<tr>
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<td>Δ Height × chronological age</td>
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<td></td>
<td>Δ Height (cm/year)</td>
<td>1.75 (0.55 to 5.59)</td>
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<td>Δ Height × skeletal age</td>
<td>0.97 (0.89 to 1.05)</td>
<td>0.44</td>
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<td>Hours per week</td>
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<td>Δ Body mass (kg/year)</td>
<td>1.74 (0.40 to 7.65)</td>
<td>0.46</td>
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<td>0.51</td>
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<tr>
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<td>0.97 (0.86 to 1.08)</td>
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</tr>
<tr>
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<td>Hours per week</td>
<td>1.23 (1.01 to 1.51)</td>
<td>0.044</td>
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<td>Δ Body mass (kg/year)</td>
<td>0.82 (0.32 to 2.11)</td>
<td>0.68</td>
</tr>
<tr>
<td></td>
<td>Skeletal age</td>
<td>0.78 (0.50 to 1.20)</td>
<td>0.25</td>
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<tr>
<td></td>
<td>Δ Body mass × skeletal age</td>
<td>1.02 (0.95 to 1.09)</td>
<td>0.58</td>
</tr>
<tr>
<td></td>
<td>Hours per week</td>
<td>1.23 (1.00 to 1.52)</td>
<td>0.05</td>
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</table>

The associations between changes in height and injuries have been reported in Dutch, Belgian, and English high-level football players [11–14]. Although these studies suggest a growth-injury relationship, different analytical approaches and broad injury categories make definite conclusions difficult. To improve our understanding of growth as an injury risk factor, we included bone tissue and physis injuries as specific outcomes, based on the assumption that they are more closely aligned with the suggested underlying mechanisms. Impor-
tantly, these injuries are among the most common and burden-some in U13–U15 age group players [21–23], making them a pri-

ority for targeted injury reduction programs. The mean age of our 
sample (13.5 years) was also close to the expected age at PHV (13.6 
years) in this specific academy population [30], which has been 
highlighted as a vulnerable phase for football players [7–10]. Still, 
we did not find any practically relevant effects of changes in height 
or body mass over an academy semester, with only one significant 
association suggesting an increased risk of sudden onset injuries 
with greater changes in body mass in the model adjusting for chron-

ological age.

The absence of observed effects in our study may be explained by 
the methodological approach, using isolated and pre-deter-

mined growth intervals (i.e., Start-Mid and Mid-End of a season). The 
duration of growth intervals reflected recommendations for 
assessing growth velocity (i.e., every 3 to 4 months [33]), but they 
do not necessarily capture the periods of most rapid growth with-
in an individual’s growth process. They may also not be frequent 

enough to identify shorter bursts of growth, which could be of in-

terest [32]. Capturing data on a more frequent basis is, however, 
associated with greater variance in the estimated growth velocities [40], 
and recommendations to focus on long-term tracking of anthropo-
metric data therefore seem reasonable [33]. This approach was taken 
by Monasterio et al. [10, 15], who calculated full growth curves. 
They did, unfortunately, not have individual training and match data avail-

able and growth curves could only be calculated for around 10% 
of their initial sample, highlighting the logistical challenges associated 
with accurately tracking growth, injuries, and individual exposure over 
a sufficient duration in applied academy settings.

Practically relevant effects of age, but not maturity, 
on injury risk

Studies have indicated a changing injury pattern with age, where 
physeal or “growth-related” injuries are more common in younger 
age groups [21–23]. This may be attributed, in part, to maturity 
status [18, 19, 41], with the immature skeleton representing a rel-
atively weak link in the muscle-tendon-bone chain prior to reach-
ing its mature state [42]. Consequently, similar injury mechanisms 
would result in different pathology types (e.g., apophysitis or avu-
lsions as opposed to tendinopathies or muscle strains) depending 
on a player’s maturity status [42]. These patterns provide a ration-
el for regular maturity assessments, which can be used to better 
accommodate for early, on time, and late maturing players within 
an age group, who may be prone to different injury types at differ-
ent locations.

In our sample, higher chronological age was related to a practi-
cially relevant increased risk of overall and sudden onset injuries, 
with point estimates suggesting moderate to large harmful effects 
on these injury outcomes. This is in line with our earlier study, which 
demonstrated increased injury incidence in older age groups in this 
academy [21]. Although large variations in skeletal age were ob-
ined within age groups (e.g., a six-year range in skeletal age be-
 tween players in the U14 age group), we did not detect any practi-
cially relevant effects of maturity on injury risk. A significant asso-
ciation was found between older skeletal age and sudden onset 
injury risk; still, we urge caution when interpreting this, as we could 
not demonstrate consistent associations across models. This may 

be due to a relatively small number of events for the number of vari-
ables included in our models, and studies including larger samples 
are needed to better understand these relationships.

Weekly load may affect the risk of gradual onset and 
bone tissue injuries

Football load (operationalized as hours per week in this study) was 
a significant covariate in all model combinations for gradual onset 
and bone injuries, and in one model for physis injuries. Again, these 
associations could not be classified as practically relevant, and a 
causal relationship cannot be established based on our study de-
sign. Youth-specific consensus statements have previously high-
lighted training load as a risk factor for these injury types [43, 44], 
and studies in high-level youth football have related weekly dura-
tion, cumulative absolute training loads over three and four weeks, 
and greater week-to-week changes in exposure to injury risk 
[14, 45, 46]. While their findings are inconsistent and our results 
do not provide definite answers, it does appear sensible to focus 
on careful progression, diverse and variable movement exposure, 
careful scheduling of total load, and allowing for sufficient rest and 
recovery to ensure positive adaptations of training while minimiz-
ing the risk of injury in youth settings [3, 43, 44]. Future studies may 
be able to incorporate more detailed measures of load in their anal-
yses to determine whether training load or athlete responses (e.g., 
physiological, perceptual) are associated with injury occurrence 
during specific phases of the growth and maturation process.

Methodological considerations

We addressed several limitations from previous studies on growth, 
maturity, and injury risk with our models accounting for 
growth × age/maturity interaction, repeated player-seasons, and 
daily individual football exposure, including more detailed injury 
outcomes recorded and verified by on-site medical staff. It is indeed 
rare that youth football teams have designated medical staff pre-
ent at each session, and that the detailed injury data they captured 
can be merged with accurate individual football exposure and skel-
etal age assessments. Still, there are limitations with our study de-
sign that should be considered when interpreting our results.

First, our growth and maturity data were primarily collected for 
clinical and applied purposes, which led to a substantial number of incomplete assessments (> Fig. 1), presumably at random and not 
related to injury occurrence. Using data collected for ongoing mon-
itoring purposes also introduces the possibility that training con-
tent could have been adjusted based on the data collected. How-
ever, we do not believe that these assessments impacted signifi-
cantly on training content during the data collection period, as 
 systematic and contextualized reports were not available to coach-
es at the time. The exploratory nature of our study, involving a ret-
rospective analysis, also precluded any formal a priori sample size 
estimation relevant to a mixed-effects modeling framework. Fu-
ture studies could improve in this area by employing strict data col-
lection routines to avoid missing data and should attempt to in-
clude a larger and younger sample of players than we were able to 
in our study, to cover the entire growth process and allow for with-
in-subject analyses.

Second, including skeletal age as our main indicator of biologi-
cal maturity is arguably a strength as it is considered a precise and
reliable measure and can be used across the maturation process [4, 16]. However, we recognize its limitations. The assessment requires equipment and expertise (and is therefore costly), and importantly involves low-dose radiation (although this has been described as almost negligible [47]). It is therefore considered an invasive measure that is not feasible to include in most academy settings [3], reducing the direct applicability of our findings to practice. Furthermore, the ossification of the hand-wrist complex does not necessarily represent the maturity status of bones in other locations. Future studies may be able to use radiation-free alternatives, such as magnetic resonance imaging or ultrasound [47], to relate skeletal maturity of multiple body parts to injuries in surrounding tissues.

Finally, although we could expand on findings from previous studies by using injury outcomes that are more closely aligned with the proposed mechanisms for a growth-maturity-injury relationship, the use of a time-loss definition limited our ability to capture injuries that did not affect participation [28]. Future studies could improve in this area by using athlete-reported measures with a broader, any-complaint definition. Readers should also be cognizant that a study like ours may not be representative for other contexts (e.g., different geographical regions, training environments, or match schedules/formats) and addresses but a few of many potential risk factors for injury, which are thought to be both multifactorial, dynamic, and complex [48].

Conclusion

Our main research question was whether changes in height or body mass between assessments at the start, middle, and end of a football season affect the risk of injury in male academy football players. After accounting for age or skeletal maturity, growth × age/maturity interaction effects, and football exposure, no such associations were considered practically relevant based on our pre-defined thresholds. We do, however, report practically relevant harmful effects of older age on overall and sudden-onset injury risk. Additionally, significant but not practically relevant associations were observed between greater changes in body mass and older skeletal age on sudden onset injury risk, and higher weekly load on gradual onset, bone tissue, and physis injuries. Researchers should strive to establish robust surveillance systems that can include larger and more diverse samples (athletes of both genders from multiple settings) and capture the whole growth and maturity process (longitudinal tracking starting from pre-adolescence) alongside reliable collection of injury and exposure data (following consensus procedures and universal classification systems), to better understand the relationship between growth, maturation, and injury risk.

Acknowledgements

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Conflict of Interest

The authors declare that they have no conflict of interest.

References


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