

A Predictive Model to Estimate Knee-Abduction Moment: Implications for Development of a Clinically Applicable Patellofemoral Pain Screening Tool in Female Athletes

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Context: Prospective measures of high external knee-abduction moment (KAM) during landing identify female athletes at increased risk of patellofemoral pain (PFP). A clinically applicable screening protocol is needed.

Objective: To identify biomechanical laboratory measures that would accurately quantify KAM loads during landing that predict increased risk of PFP in female athletes and clinical correlates to laboratory-based measures of increased KAM status for use in a clinical PFP injury-risk prediction algorithm. We hypothesized that we could identify clinical correlates that combine to accurately determine increased KAM associated with an increased risk of developing PFP.

Design: Descriptive laboratory study.

Setting: Biomechanical laboratory.

Patients or Other Participants: Adolescent female basketball and soccer players (n = 698) from a single-county public school district.

Main Outcome Measure(s): We conducted tests of anthropometrics, maturation, laxity, flexibility, strength, and landing biomechanics before each competitive season. Pearson correlation and linear and logistic regression modeling were used to examine high KAM (>15.4 Nm) compared with normal KAM as a surrogate for PFP injury risk.

Results: The multivariable logistic regression model that used the variables peak knee-abduction angle, center-of-mass height, and hip rotational moment excursion predicted KAM associated with PFP risk (>15.4 Nm of KAM) with 92% sensitivity and 74% specificity and a C statistic of 0.93. The multivariate linear regression model that included the same predictors accounted for 70% of the variance in KAM. We identified clinical correlates to laboratory measures that combined to predict high KAM with 92% sensitivity and 47% specificity. The clinical prediction algorithm, including knee-valgus motion (odds ratio [OR] = 1.46, 95% confidence interval [CI] = 1.31, 1.63), center-of-mass height (OR = 1.21, 95% CI = 1.15, 1.26), and hamstrings strength/body fat percentage (OR = 1.80, 95% CI = 1.02, 3.16) predicted high KAM with a C statistic of 0.80.

Conclusions: Clinical correlates to laboratory-measured biomechanics associated with an increased risk of PFP yielded a highly sensitive model to predict increased KAM status. This screening algorithm consisting of a standard camcorder, physician scale for mass, and handheld dynamometer may be used to identify athletes at increased risk of PFP.

Key Words: high-risk biomechanics, patellofemoral risk factors, targeted neuromuscular training, knee injury prevention, assessment tools

Key Points

- With readily available clinical measures, clinicians may use the clinical algorithm to identify athletes at increased risk of developing patellofemoral pain.
- Once higher-risk athletes are identified, clinicians can implement targeted intervention programs to correct deficits and potentially reduce the risk of patellofemoral pain.

Patellofemoral dysfunction and the resultant patellofemoral pain (PFP) affect up to 30% of people aged 13 to 19 years.¹ Three-quarters of patients with PFP dysfunction are limited in their recreational activities or have ceased physical activity altogether.² A significant sex disparity is associated with the development of PFP: young females are affected more often than their male counterparts.³ Altered or reduced motor control during physical activities, which results in excessive frontal and transverse knee-joint motion and load in females, may contribute to the development of patellofemoral dysfunction.⁴

In a recent investigation,⁴ middle and high school female athletes ($n = 240$) were evaluated by a physician for PFP and for landing biomechanics before their basketball season and monitored for athlete-exposures and PFP injury during their competitive seasons. The cumulative incidence rate for the development of PFP was 1.09 per 1000 athlete-exposures. Athletes with newly diagnosed PFP demonstrated increased knee-abduction moment (KAM) at initial contact on the most symptomatic side. Regression analysis from the same investigation indicated that the PFP risk was increased in athletes who demonstrated > 15.4 Nm of knee abduction during landing.⁵ Targeted preseason neuromuscular-training interventions for those who have underlying mechanics associated with a high KAM risk factor may reduce PFP incidence. Predictive models of KAM magnitude based on laboratory tools showed that deficits related to frontal- and sagittal-plane knee mechanics were strongly related to increased anterior cruciate ligament injury risk in those with higher magnitudes of KAM.⁶ The current gap in our knowledge is the underlying biomechanical predictors for KAM that may be most closely related to the development of PFP. In addition, we do not currently know if clinic-based measurements are adequate to identify athletes at increased risk for developing PFP.

Our primary purpose was to identify biomechanical laboratory measures that accurately predict KAM loads during landing and that predict an elevated risk of PFP in young female athletes.⁴ The secondary purpose was to identify clinical correlates to laboratory-based measures of increased KAM status for use in a clinical PFP injury-risk prediction algorithm. Our hypothesis was that clinically obtainable measures derived from the highly predictive laboratory-based models would be accurate in identifying increased KAM status in female athletes at greater risk for PFP.

METHODS

Participants

Between 2004 and 2008, all 6th- through 12th-grade female basketball and soccer players were recruited from a county public school district (5 middle schools, 3 high schools) to participate in a prospective longitudinal study. The recruited schools consisted of 6 high school and 15 middle school basketball teams and 7 high school and 3 middle school soccer teams. From the identified teams, first-time visits of 744 participants were included in the current analyses; 46 volunteers were excluded from the study because either they did not complete biomechanical testing ($n = 3$) or we found errors in variables used to calculate KAM measures (eg, foot not entirely located on

force platform, markers excluded from camera view; $n = 43$). Thus, 698 participants were included in the final study analyses (age = 13.9 ± 2.4 years, height = 159.3 ± 8.6 cm, body mass = 54.0 ± 12.5 kg, maturational status = 17.2% prepubertal, 29.9% pubertal, and 52.8% postpubertal).

Procedures

Before data collection, we obtained study approval from the institutional review board and parental consent and athlete assent. Participants were tested before the start of their basketball or soccer competitive season. The testing consisted of a medical history, knee examination, anthropometric measurements, maturational estimates, laxity and flexibility measurements, dynamic strength measurements, lower extremity segment length and alignment measurements, and landing biomechanical analysis. The screening methods have been previously reported and provided good reliability.^{6,7}

Anthropometric Measurements. Height was measured using a stadiometer with the participants barefoot. We conducted a static motion-analysis trial to calculate standing anatomic alignment measures (detailed later) that were considered in the prediction algorithm. Body mass was measured on a calibrated balance scale. Body mass index Z-score was calculated using SAS (gc-calculate-BIV.sas; available on the Centers for Disease Control and Prevention Web site: <http://www.cdc.gov/nccdphp/dnpao/growthcharts/resources/sas.htm>).

Maturational Assessment. We used a modified Pubertal Maturational Observational Scale (PMOS) consisting of parental questionnaires and observations to classify participants into 1 of 3 pubertal categories (prepubertal, pubertal, or postpubertal). A single investigator used the PMOS to assess and classify each participant into a pubertal category.⁸ The PMOS scale has high reliability and can differentiate among pubertal stages based on indicators of adolescent growth, breast development, menstruation status, axillary and leg hair presence, muscular development, presence of acne, and evidence of sweating during physical activities.

Laxity and Flexibility Measurements. Generalized joint laxity was identified by fifth-finger hyperextension greater than 90° , elbow hyperextension greater than 10° , wrist and thumb to forearm opposition, knee hyperextension greater than 10° , and palms to floor.⁹ Tibiofemoral translation was quantified using the CompuKT knee arthrometer (MEDmetric Corporation, San Diego, CA) to measure total anterior-posterior displacement of the tibia relative to the secured femur. During the measurement, each leg was placed on the adjustable thigh support with the knee stabilized at 20° to 35° of knee flexion. The arthrometer was secured to the shank such that the patellar sensor pad rested on the patella with the knee joint-line reference mark on the arthrometer aligned with the participant's joint line. The ankle and foot were stabilized to limit leg rotation. The tester provided anterior and posterior pressure (± 134 N) to a plane perpendicular to the long axis of the tibia. Total displacement (mm) was plotted on the computer and recorded. Side-to-side differences in knee laxity were calculated from the absolute difference in total anterior-posterior tibial translation between the dominant and nondominant knees.⁹

Dynamic Strength Measurements. Isokinetic knee flexion-extension (concentric-concentric muscle action) strength was measured with the participant seated on the dynamometer and the trunk perpendicular to the floor, the hip flexed to 90°, and the knee flexed to 90°. Before we collected each set of data, the participant performed a warm-up set consisting of 5 submaximal knee flexion-extension repetitions for each leg at 300°/s. The test session consisted of 10 knee flexion-extension repetitions for each leg at 300°/s. Peak flexion and extension torques were recorded (ft·lb).¹⁰ We measured concentric hip-abduction strength with the participant standing erect, fully supported, with a stabilization strap around the pelvis and her hands gripping a stable hand rest. The test leg was positioned lateral to the opposite leg in 0° of hip and knee flexion. The axis of hip abduction-adduction was aligned with the axis of rotation of the dynamometer. The resistance pad was affixed to the participant's leg, immediately proximal to the knee joint. Each participant was provided with instructions and allowed to execute 5 submaximal warm-up hip abduction-adduction movements at the test speed. The warm-up was immediately followed by the test session, which consisted of 5 maximal-effort hip-abduction maneuvers with passive adduction repetitions at 120°/s.¹¹ These speeds approximate actual hip abduction-adduction velocities quantified kinematically during high-risk cutting tasks. The initial test leg was alternated between participants to control for a side learning effect. Peak hip-abduction torques were recorded, and all examinations were performed by a single investigator.¹²

Lower Extremity Segment Length and Alignment Measurements. Before biomechanical testing, we conducted a static standing trial in which the participant was instructed to stand still with foot placement standardized to the laboratory coordinate system. The static standing trial was used to calculate lower extremity segment lengths as the estimated distance between the proximal and distal joint center (eg, the thigh segment distance was equal to the distance between the hip-joint center and the knee-joint center). From the mass and inertial properties for each segment (based on sex-specific values from de Leva¹³), we calculated the height of the center of mass (COM).

Landing Biomechanical Analysis. Three-dimensional hip, knee, and ankle kinematic and kinetic data were quantified for the contact phase of 3 repetitions of a drop vertical-jump (DVJ) task. A single investigator placed 37 retroreflective markers on each participant: the sacrum, left posterior-superior iliac spine, sternum, and bilaterally on the shoulder, elbow, wrist, anterior-superior iliac spine, greater trochanter, mid-thigh, medial and lateral knee, tibial tubercle, mid-shank, distal shank, medial and lateral ankle, heel, dorsal surface of the midfoot, lateral foot (fifth metatarsal), and toe (between the second and third metatarsals). As for the static trial, the participant was instructed to stand still with foot placement standardized to the laboratory coordinate system. This static measurement was used as each person's reference alignment; subsequent kinematic measures were referenced in relation to this position.⁷ For the DVJ, the participant stood on top of a box (31-cm high) with her feet positioned 35 cm apart. She was instructed to drop directly down off the box and

immediately perform a maximum vertical jump, raising both arms as if jumping for a basketball rebound.⁷

A single investigator collected all trial data with EVA_{RT} (version 4; Motion Analysis Corporation, Santa Rosa, CA) using a system consisting of 10 digital cameras (Eagle cameras; Motion Analysis Corporation) positioned in the laboratory and sampled at 240 Hz. Before data collection, we calibrated the motion-analysis system based on the manufacturer's recommendation. Two force platforms (Triaxial; Advanced Mechanical Technology, Inc, Watertown, MA) were sampled at 1200 Hz and time synchronized with the motion-analysis system. The force platforms were embedded into the floor and positioned 8 cm apart so that each foot would contact a different platform during the stance phase of the DVJ.⁷

After data collection, we further analyzed the motion and force data in Visual3D (version 4.0; C-Motion, Inc, Germantown, MD). Net external knee moments were described in this article and represent the external load on the joint. Lower extremity kinetics and kinematics were calculated during the deceleration phase of landing from the stance phase of the DVJ. The *deceleration phase* was operationally defined from initial contact (when vertical ground reaction force first exceeded 10 N) to the lowest vertical position of the body COM. The left-sided data were used for statistical analysis. The described landing and biomechanical landing analysis techniques have demonstrated reliable measurements.⁷

Statistical Analyses

Data were exported to SPSS (version 16.0; IBM SPSS Statistics, Armonk, NY) and SAS (version 9.1; SAS Institute Inc, Cary, NC) for statistical analysis. Because of the nearly 2500 potential biomechanical, anthropometric, and strength variables available for inclusion in the initial prediction model, we used Pearson correlation coefficients to assist in the initial screening of independent variables, which enabled us to remove nonsignificant correlates of the dependent variable; that is, associations with values of $P > .05$ were not included in the regression model. Imputation of missing data observed for the remaining independent variables was performed using Solas (version 3.0; Statistical Solutions, Saugus, MA). The imputation method was based on ordinary least-squares regression, using age and body mass index as covariates, and was necessary for less than 2% of the observations on any independent variable. Measures of pubertal status were coded into 2 dummy variables with postpubertal status as the referent group.

To further reduce the number of independent variables, we used SAS exploratory cluster analysis techniques to identify and group variables with shared variance into meaningful taxonomies. Using this technique, 20 clusters were identified for the initial laboratory-based prediction model. The next step involved regression analyses using the independent variables from each cluster separately. We initially ranked the variables within each cluster based on their clinical relevance and thus appropriateness for inclusion in the regression models. No more than the top 3 ranked variables from each cluster were included in these multivariable regression analyses.

For the initial step in model development, we performed a multivariable receiver operating curve analysis to

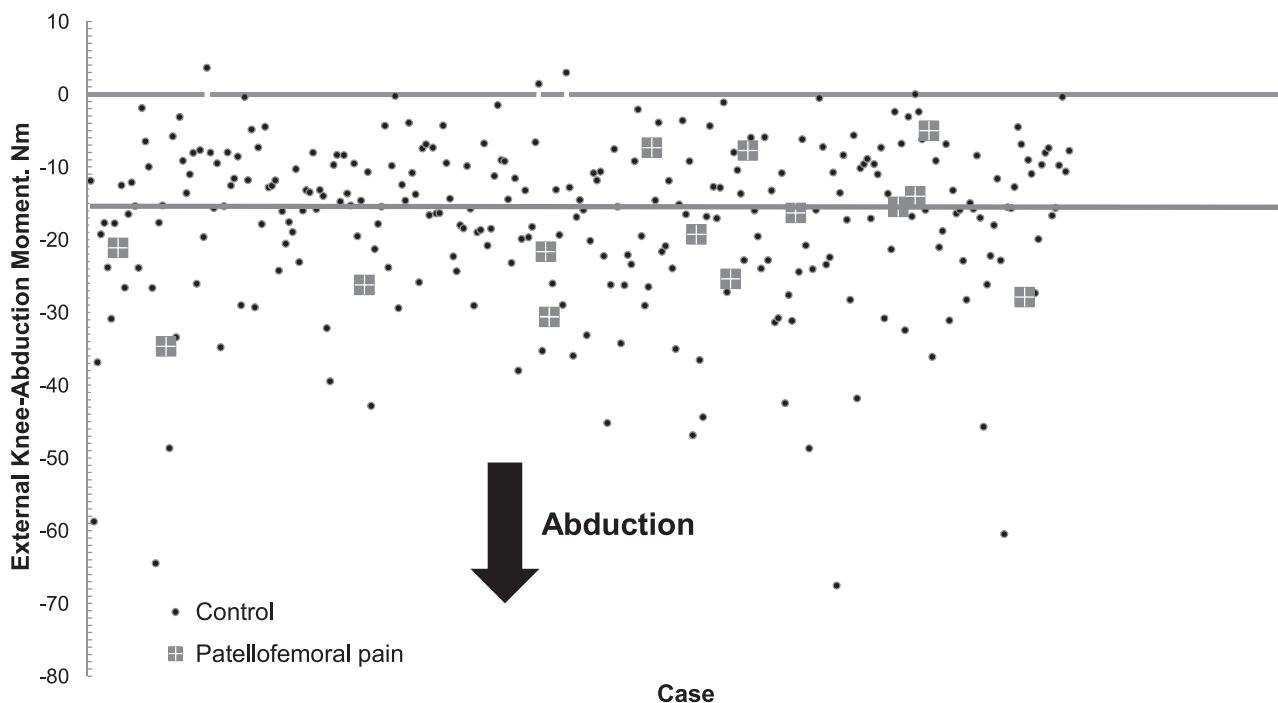


Figure 1. Prospective knee-abduction moment data from participants with or without patellofemoral pain that were used to define the cut score with maximal sensitivity and specificity.

determine a cut score that would provide the maximal sensitivity and specificity for predicting PFP injury risk during a DVJ (Figure 1).^{4,5} The cut point from the receiver operating characteristic analysis used to classify the dependent variable status was >15.4 Nm of KAM. Using this classification, participants were categorized into a dichotomous (high KAM: *yes* or *no*) outcome variable. As previously described, all potential predictor variables remaining after cluster variable reduction were introduced into a logistic regression model to predict high (>15.4 Nm) versus low (≤ 15.4 Nm) KAM.

For the final step in laboratory-based model development and validation, we used a multivariable linear regression to evaluate the chosen prediction model for the continuous outcome of external knee-abduction load. The final model was tested for validity based on examination of the variance inflation factor to quantify the degree of multicollinearity. Jack-knife residuals were plotted and Cook distance statistics inspected to identify potential outliers. Finally, the residuals were plotted and the Kolmogorov-Smirnov statistic was used to test for possible deviations from normality.

We calculated Pearson correlation coefficients to assist in the initial screening of surrogate clinical predictor variables by selecting significant correlates ($P < .01$) of the independent laboratory-based variables used in a previous model to predict continuous KAM and the dichotomized high KAM (KAM > 15.4 Nm) status. The remaining 28 potential independent variables available for consideration in the clinical prediction model are presented in Table 1. For the final step in the clinical model development, multivariable logistic regression with a backward-elimination strategy was conducted. The multivariable logistic regression model was estimated using a logit link. An alpha level of .05 was set for all models. The multivariable

regression model's predictive accuracy was quantified with the use of the C statistic, which corresponds to the area under the receiver operating characteristic curve. A nomogram was produced from the secondary classification model to allow predictions in individual athletes.¹⁴ The clinical nomogram was developed using R Project for Statistical Computing software (Free Software Foundation, Inc, downloaded 2009, Boston, MA). The R Project software is available as free software under the terms of the Free Software Foundation's GNU General Public License in source code form.

RESULTS

Laboratory-Based Model

The multivariable logistic regression model that used the variables (1) peak knee-abduction angle, (2) COM height, and (3) hip-rotation moment excursion (maximum range of hip rotational torque: ie, absolute value of minimum–maximum value) predicted KAM knee load associated with increased PFP risk (>15.4 Nm of KAM) with 92% sensitivity, 74% specificity, and a C statistic of 0.93. The multivariable linear regression model that included the same independent predictors accounted for 70% of the variance in KAM during landing.

Clinic-Based Model

We identified significantly related clinical correlates to laboratory-based measures that combined to predict increased KAM status with 92% sensitivity and 46% specificity. The clinical prediction algorithm, including knee-valgus excursion (odds ratio [OR] = 1.46, 95% confidence interval [CI] = 1.31, 1.63), COM height (OR = 1.21, 95% CI = 1.15, 1.26), and ratio of body fat

Table 1. Factors Considered Independent Clinical Predictors for Developing Patellofemoral Pain

Anterior-posterior tibial translation @20 lb (9.07 kg), mm
Anterior-posterior tibial translation @30 lb (13.6 kg), mm
Body mass index, mass/height ²
Body mass index Z score, no. of SDs
Body mass, kg
Center-of-mass height, cm
Generalized joint laxity, Beighton-Horan score
Hamstrings flexibility, °
Height, cm
Isokinetic hip-abduction strength, ft•lb
Isokinetic knee-extension to knee-flexion strength, ratio
Isokinetic knee-extension strength, ft•lb
Isokinetic knee-flexion strength to body fat percentage, ratio
Isokinetic knee-flexion strength, ft•lb
Knee-flexion range of motion during stance phase of drop vertical jump, °
Knee-valgus excursion, cm
Normalized isokinetic hip-abduction strength, Nm•kg
Normalized isokinetic knee-extension strength, Nm•kg
Normalized isokinetic knee-flexion strength, Nm•kg
Pubertal status
Relative body composition, fat %
Shoe size
Standing knee-abduction angle, °
Standing quadriceps angle, °
Tibia length, cm
Trunk segment length, cm
Vertical jump height, cm

percentage to knee-flexion strength (OR = 1.80; 95% CI = 1.02, 3.16) predicted high KAM status with a C statistic of 0.80. Mean values for the univariate clinical predictors are presented in Table 2.

A predictive nomogram developed from the analysis described earlier that can be used to predict high KAM (>15.4 Nm) based on COM height, knee-valgus excursion, and ratio of body fat percentage to knee-flexion strength (ft•lbs) is presented in Figure 2.

DISCUSSION

Over the past 4 decades, a large sex disparity in knee injuries and knee pain has been reported among young female athletes, and this divergence does not appear to be decreasing.¹⁵ The current standards aimed at prevention and treatment of PFP are based on retrospective and case-control biomechanical investigations that attempted to extrapolate the demonstrated altered neuromuscular strategies in patients with active symptomatic PFP into potential predisposing mechanisms.^{16,17} However, the retrospective nature of the prior biomechanical and strength analyses of symptomatic patients' data has limited the ability to determine if the measured deficits contribute to the development of PFP or if they present as sequelae of the disorder in its natural course. The proposed clinical algorithm is innovative, as it is focused on prospectively identifying predisposing risk factors that may lead to PFP in young female athletes. The proposed mechanistic-based clinical algorithm may effectively position us to apply patient-specific interventions before PFP develops and ultimately prevent the underlying mechanisms from manifesting. The development of the clinical screening tool from populations captured prospectively creates the

Table 2. Descriptive Statistics for Independent Clinical Prediction Variables

Independent Predictor	Mean ± SD
Knee-valgus excursion, cm	3.99 ± 2.14
Center-of-mass height, cm	89.10 ± 5.12
Isokinetic knee-flexion strength to body fat percentage, ratio	0.76 ± 0.33

potential for a paradigm shift in prevention and treatment strategies for PFP.

Mechanistic Connection Between Rapid Increases in COM

The current findings indicate that laboratory-based measures of peak knee-abduction angle, COM height, and hip-rotation moment excursion (maximum range of hip-rotation torque) combined to accurately predict knee load associated with elevated PFP risk.⁴ Rapid growth during maturation results in concomitant increases in the height of the COM, making neuromuscular control of the trunk more challenging. In addition, increased body mass with longer joint levers initiates greater joint forces that are more difficult to balance and dampen at the lower extremity joints during high-velocity maneuvers.^{18,19} Trunk stability after rapid growth and increased COM height likely depends on the ability of the hip to control the trunk in response to forces generated from distal body segments and from unexpected perturbations.^{20,21}

If the hip musculature is unable to control the position of the COM during cutting and landing, uncontrolled lateral trunk motion may increase knee-abduction motion and torque through mechanical (lateral ground reaction force motion) and neuromuscular (increased hip-adductor torque) mechanisms.^{22,23} Maturing females do not demonstrate sufficient neuromuscular adaptations (eg, deficient eccentric tension in the hip-abductor musculature is responsible for increased adduction of the hip joint) to match the increased demands created by structural and inertial changes during pubertal development.²¹ As they mature and COM height increases, females tend to use greater frontal-plane force-absorption strategies rather than a sagittal-plane control strategy for the lower extremity.^{24,25} The knee, especially the patellofemoral joint, functions optimally with sagittal-plane mechanics as the large muscles of the lower extremity that control frontal-plane trunk, hip, and knee motion or torque absorb and dissipate force most effectively and efficiently in the sagittal and not the frontal plane.^{4,21,26}

Ipsilateral trunk lean may be a sign of weak hip abductors or lack of motor control of the trunk as it moves the COM closer to the stance limb to reduce demand on the weak abductors,²⁷ and deficits in neuromuscular control of the hip may exacerbate laterally directed trunk motions.²¹ Considering that the trunk accounts for more than half of the body's mass, increased height of COM, combined with reduced hip strength and deficient recruitment of the hip musculature (ie, hip-abductor muscles), likely increases lateral displacement of the location at which the vertical ground reaction force acts on the lower extremity joints. This lateral displacement of the vertical ground reaction force vector can disrupt the resultant internal response of an

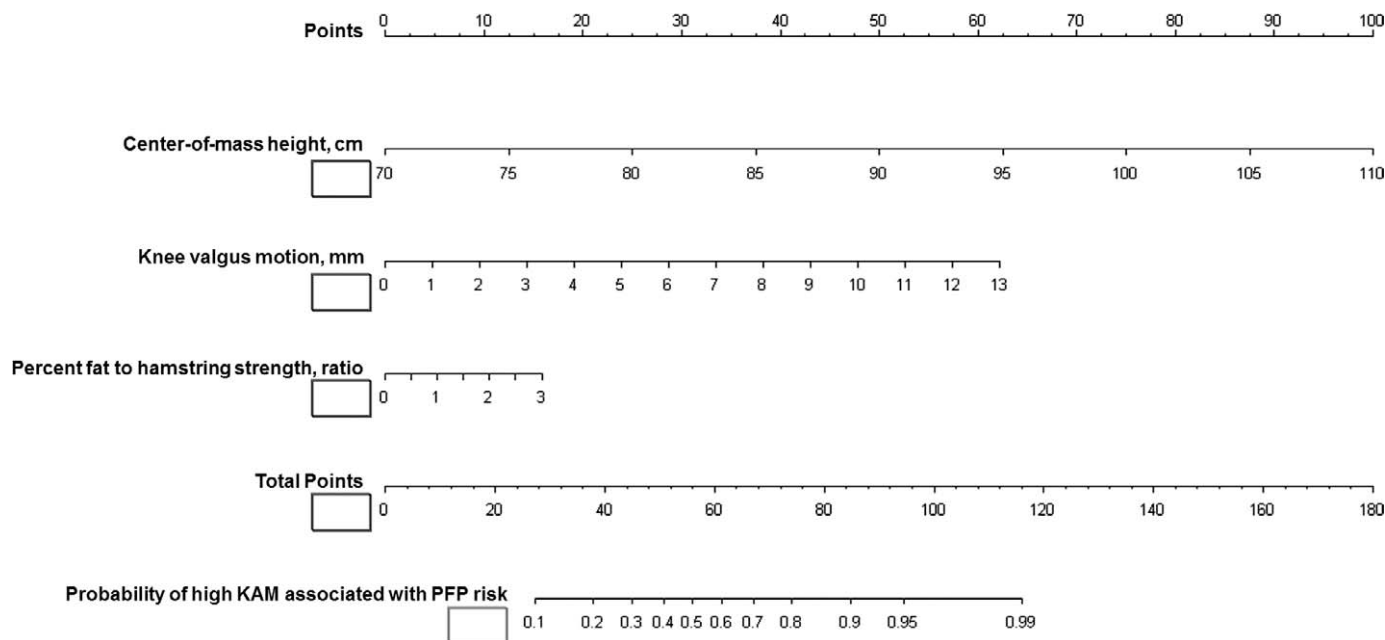


Figure 2. The nomogram code generates an equally distributed, segmented line representing standardized measurable units for each clinical predictor variable of knee-abduction moment. The magnitude of each intrasegment distance is in direct proportion to that individual variable's strength of association (β coefficient) to the predicted outcome as determined by the combined multivariable logistic regression equation. To use the prediction nomogram, a straight edge can be placed vertically so that it touches the designated variable on the axis for each predictor value, and the recorded value for each of the 3 predictors is used to mark points on the axis at the top of the diagram. All of the recorded points measured using this method can then be summed and this value located on the total points axis. A straight edge can then be used to bisect the total points line to the probability line, which gives the probability that the athlete demonstrated high knee-abduction moment (>15.4 Nm of knee abduction) during the drop vertical jump based on the predictive variables.

equal and opposite increase in the counterbalancing hip-adductor torque.^{27,28} Preliminary data from our large prospective cohort of young female athletes indicate that excessive lateral trunk motion, external hip-adduction moment induced by ground reaction force, and disrupted knee mechanics are associated with increased PFP risk and are highly correlated during dynamic movements.²¹ Decreased hip-abductor strength and increased hip adduction during dynamic motion and recruitment can disrupt normal knee kinetics, altering frontal-plane knee loads and patellofemoral mechanics that ultimately increase the risk for PFP development.^{4,23,29} Cumulatively, this evidence drives our central hypothesis that predisposing morphologic characteristics associated with growth and maturation, in concert with a lack of fully developed lower extremity hip-stabilizing mechanisms, initiate abnormal patellofemoral joint mechanics that lead to the onset of PFP in maturing young females. To guide our clinical model development, we first determined the multivariable logistic regression model that used the variables (1) peak knee-abduction angle, (2) COM height, and (3) hip-rotation moment excursion (maximum range of hip-rotation torque) to provide a highly accurate prediction of the KAM risk factor. From this laboratory-based model, we identified significant clinical correlates to the laboratory-based predictors that we could evaluate in the proposed clinical prediction model.

Clinic-Based Frontal-Plane Knee Motion

Altered or decreased neuromuscular control during the execution of sports movements that result in excessive resultant frontal-plane knee-joint motion and load appeared

to increase the risk of acute anterior cruciate ligament injury in female athletes.²³ These same factors may also be associated with the development of PFP. Army recruits who landed from a jump with a combination of reduced knee flexion, increased out-of-plane hip rotation, and frontal-plane foot laxity were at increased risk for developing PFP.³⁰ Previous authors^{23,26} have suggested that abnormal frontal-plane kinematics and moments that are associated with acute injury may also be related to PFP development in young athletes, but this relationship has not been prospectively examined in dynamic sport populations who develop PFP. At the small knee-flexion angles (19° to 22°) commonly found in female athletes when initiating a landing, abnormal frontal-plane patellofemoral joint kinematics were evident in those with PFP.^{17,24} Specifically, at 19° of knee flexion, patients with PFP presented with a laterally aligned patella, which was also associated with increased knee abduction.¹⁷ Cumulatively, the current evidence indicates that excessive knee valgus, especially at small knee-flexion angles similar to those found at initial contact in a jump landing, should be evaluated to determine its contribution to PFP onset.

In the current data set, 2-dimensional knee-valgus excursion within the frontal plane was a strong discriminator between the dependent variable of high (>15.4 Nm) versus low (≤ 15.4 Nm) KAM as a surrogate indicator of the risk for PFP (Figure 3). Neuromuscular control is necessary to avoid the potential for high KAM and is required to maintain dynamic knee stability during landing and pivoting.^{31,32} When the mechanics of active muscular control are disrupted, increased knee-valgus excursions in the frontal plane may disrupt normal patellofemoral loads

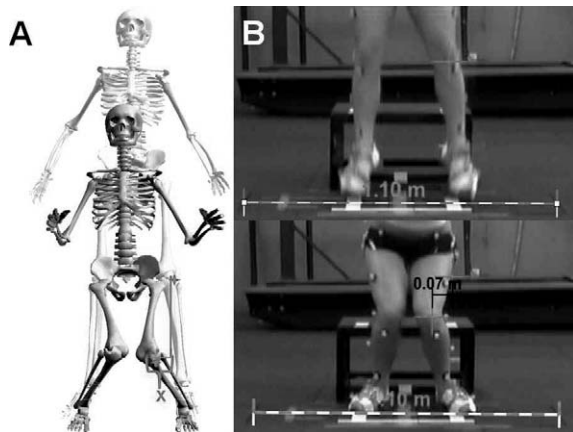


Figure 3. Kinematic frontal-plane measures captured using A, laboratory-based, and B, proposed clinical correlates. Using 2-dimensional video analysis requires the coordinate position of the knee-joint center digitized in the frontal view to be measured in the frame before initial contact and used as the knee-valgus position X1. The coordinate position of the knee-joint center digitized in the frontal view is measured in the frame with maximum medial position and used as the knee-valgus position X2. The calibrated displacement measure between the 2 digitized knee coordinates (X2–X1) represents knee-valgus excursion during the drop vertical jump. Figure reproduced from Myer GD, Ford KR, Brent JL, Hewett TE. An integrated approach to change the outcome part I: neuromuscular screening methods to identify high ACL injury risk athletes. *J Strength Cond Res.* 2012;26(8):2265–2271. With permission from the Editor.

and stresses and increase the risk of PFP injury. Because the camcorder, a simple clinic-based measure, can accurately capture 2-dimensional motion and may identify those at risk of developing PFP, its inclusion in the developed clinic-based prediction model is warranted.³³

Clinic-Based Posterior Chain Muscular Recruitment Relative to Body Composition

Without the muscular power required to resist initial contact valgus or adequate medial knee musculature cocontraction to counteract initial contact KAM scenarios, the tibial and femoral articulating surfaces open and translate, which may allow for excessive peak KAM loads.^{34,35} Ultimately, movement patterns with high KAMs may increase the potential for an athlete to increase loads on the passive ligament structures and experience abnormal patellofemoral joint stresses.³⁶

The knee joint, which is a hinge articulation of the body's 2 longest levers, is equipped with strong active muscular restraints to adequately dampen knee-joint loads in sagittal-plane motions.³⁷ Unfortunately, female athletes at high risk for developing PFP demonstrate functional impairments (eg, excessive frontal-plane knee loads) that may limit muscular protection against the disorder. In females, hamstrings muscle activation relative to quadriceps activation does not increase, and hamstrings electromyography amplitude during increased intensity of the landing phase of a jump is low.³⁸ In addition, postpubertal females demonstrate altered hip-recruitment strategies for control of landing, with greater hip moments, higher knee-to-hip moment ratios, decreased gluteus maximus activation, increased rectus femoris activation, and greater hip-adduction angles and moments than males.²⁴

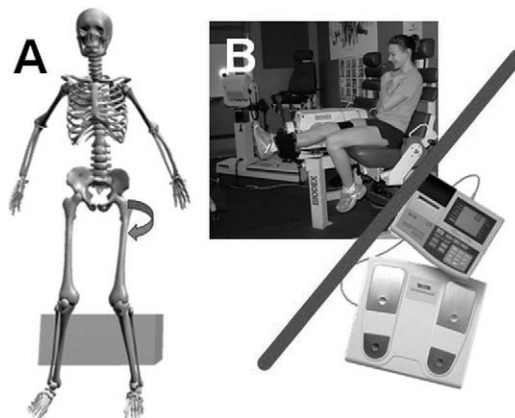


Figure 4. Hip-recruitment and -strength assessment measured using A, laboratory-based, and B, proposed clinical correlate body composition measurement that is represented by the impedance scale divided by the posterior chain strength measured (knee-flexion strength measured with a clinic-based dynamometer and recorded in ft•lb).

Increased body mass relative to height (body mass index) has been implicated as a risk factor for knee injuries; however, a recent report³⁹ indicates that body mass index may not be directly linked to an increased risk of PFP incidence in female athletes. In our prediction model, increased relative body fat was associated with an increased likelihood of classification as high KAM status and was associated with increased risk of PFP. To improve the prediction accuracy, we developed a ratio of posterior chain strength (isokinetic knee flexion) with relative body composition for use in the clinical algorithm (Figure 4). Future clinical algorithms that include closed chain measures of hip and posterior chain strength may improve the delineation of PFP risk.⁴⁰

Clinic-Based Measurement of COM Height

Beynnon et al⁴¹ reported that increased thigh length was a risk factor for knee injury in female skiers. Our results indicate that increased COM height was associated with increased propensity for KAM loads >15.4 Nm. The COM measure proposed for the clinical algorithm for PFP risk prediction (Figure 2) can be accurately captured in the clinical setting; calculations may be acquired from a standard physician's scale using the reaction-board method of calculation (Figure 5).⁴² During peak growth (height and mass) velocity in puberty, the tibia and femur grow at rapid rates in both sexes. Muscular control of the trunk becomes more challenging because of the concomitant increased height of the COM. In addition, increased body mass with longer joint levers initiates greater joint forces that are more difficult to balance and dampen at the lower extremity joints during high-velocity maneuvers.²¹ During this developmental period, male athletes naturally demonstrate a "neuromuscular spurt" (increased strength and power during maturational growth and development) to match the increased demands of growth and development but do not demonstrate the same increases in KAM observed in female athletes.^{11,43} Conversely, female athletes do not demonstrate similar neuromuscular adaptations to match the changing demands imposed by structural and inertial changes during pubertal development.^{11,21,43} Interestingly,

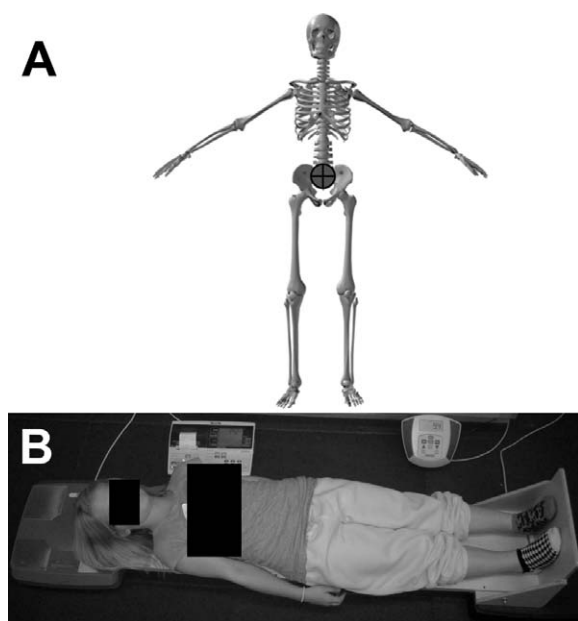


Figure 5. Center-of-mass height calculation using **A**, laboratory-based methods, and **B**, proposed clinical correlates. Reaction board center-of-mass height measurement is calculated as $x = w_2 \cdot l / (w_1 + w_2)$, where w_1 = mass (kg) measured at the scale near the feet, w_2 = mass (kg) measured at the scale near the head, l = length of the board, and x = center-of-mass height from the floor.

all new PFP cases that contributed to determining the >15.4-Nm KAM cut point for classification of high-risk status⁴ occurred in the younger middle school-aged female basketball players, which indicates that implementing targeted trunk and hip strengthening and neuromuscular training at an earlier age in high-risk athletes may help to minimize lower extremity pathomechanics and thereby reduce the risk and occurrence of PFP before these athletes participate in high school sports.

A Clinical Assessment Tool to Facilitate Target Training Strategies

Increased COM height without complementary hip strength and control has been proposed as an underlying mechanism for increased frontal-plane knee motion and load in young females as they mature.^{4,21} Decreased hip (gluteus medius and gluteus maximus) strength is related to increased knee-abduction alignment at initial contact and maximum measurement,⁴⁴ and adolescent males naturally demonstrate increased relative hip-abduction strength development during maturation, whereas females show no similar adaptive strategies.¹¹ Thus, focusing clinically on muscular performance and hip motor-control strategies to decrease dynamic valgus postures and knee-abduction loads may decrease abnormal patellofemoral loading mechanics in females during sports maneuvers.^{21,45} Neuromuscular training focused on the trunk and hip increased standing hip-abduction strength in females.¹² This training may improve the ability of females to control for the increased height of their COM and improve dynamic lower extremity alignments to reduce loads that may contribute to the onset of PFP. Accordingly, Labella et al⁴⁶ instituted a preseason neuromuscular-training program for young females and found that the prevalence of knee pain at

postseason follow-up was reduced.⁴⁶ More specifically, published work from our laboratory indicates that females categorized as high risk for lower extremity injury, based on previous coupled biomechanical and epidemiologic studies,^{4,23} may be more responsive to specially designed neuromuscular training.⁴⁷ By identifying females at greater risk for PFP, we can substantially improve prevention strategies to reduce the incidence of PFP. The use of the clinical prediction algorithm (Figure 6) may increase the efficiency of neuromuscular training if it is targeted at females who demonstrate high KAMs. Our results may also aid in the dissemination of assessment techniques required for the application of targeted neuromuscular-training interventions to high-risk populations.

Limitations

Patellofemoral pain in female athletes likely has multifactorial causes. Prior epidemiologic findings indicated that altered muscular activation or other intrinsic factors (such as anatomical, hormonal, and potentially psychological factors) may contribute to PFP onset.⁴⁸ However, we did not control for or investigate these factors in our analyses. Future investigators with larger sample sizes should aim to develop more robust PFP prediction models that include other potential contributing factors (eg, sport, training error, patellar-tracking mechanisms, hormonal measures, and potentially psychological aspects) to further elucidate risk factors for this disorder. Another possible limitation is that our analyses were limited to the strength of the relationship of KAM with the desired outcome of PFP. Continued efforts to accurately classify PFP into distinct injuries and to determine prediction models directly linked to PFP injury outcome are warranted. Finally, future prediction models that are focused on specific subclassifications of PFP diagnoses may optimize the sensitivity and specificity of prediction risk.

A further potential limitation is that based on the low risk of the treatment (neuromuscular training) for high KAM, we chose to influence the algorithm for high sensitivity of prediction for this outcome. Although an increased number of false-positive results may be predicted with efforts to maximize sensitivity, female athletes predicted to demonstrate both low and high KAM will likely improve performance as an unanticipated effect of neuromuscular training targeted at reducing injury risk. Finally, we acknowledge that the proposed algorithmic approach may have limited utility to predict injury risk during cutting, pivoting, or maneuvers not associated with landing. Future authors should aim to validate the proposed algorithm using clinical measurement techniques to determine the relationship of running and cutting injury mechanisms to PFP injury prediction. In addition, further research is warranted to delineate the most efficient and effective training methods for females who demonstrate high KAM landing mechanics to further improve the potential prophylactic effects.

CONCLUSIONS

Authors of prior prospective investigations of landing biomechanics related to increased risk of PFP have used inverse dynamics that require complex, laboratory-based, 3-dimensional kinematic and kinetic measurement tech-

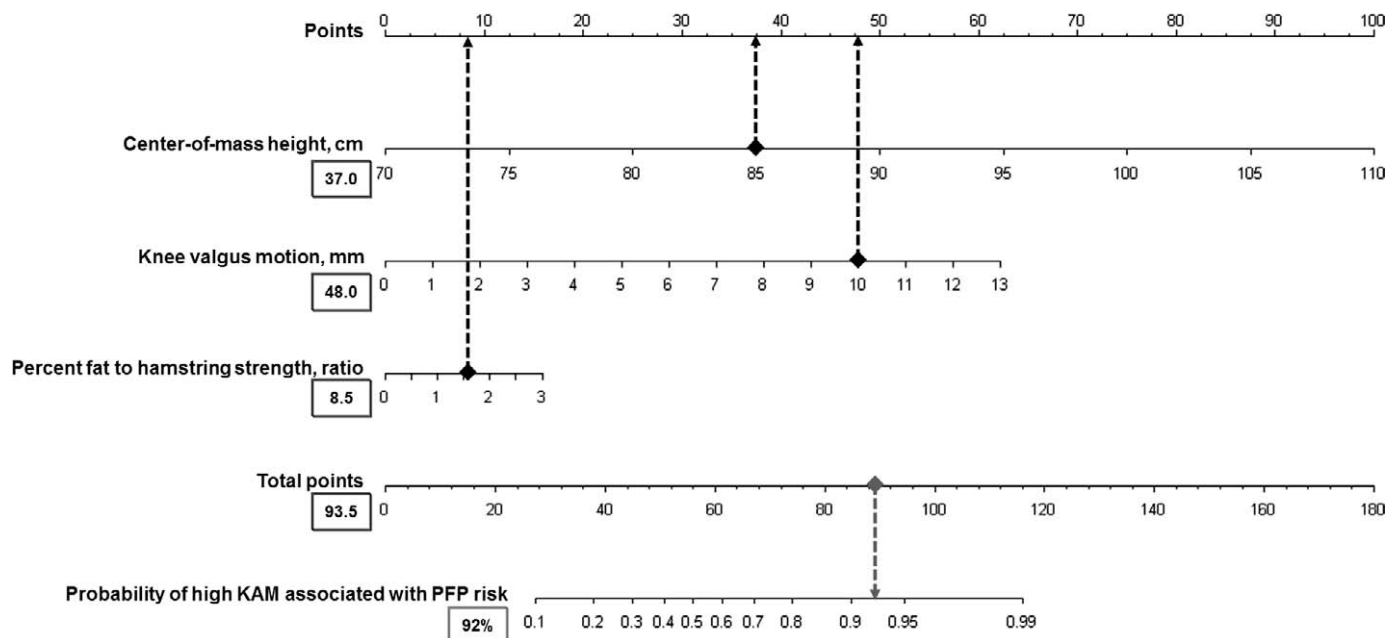


Figure 6. Completed nomogram for the representative participant (center-of-mass height = 85.0 cm, knee-valgus excursion = 10.0 cm, body fat percentage [35.0%] to hamstring strength [22.0 ft-lb] = 1.59). Based on her demonstrated measurements, this participant would have a 92% (93.5 total points) chance of exceeding the knee-abduction moment threshold associated with increased risk of developing patellofemoral pain.

niques.^{4,30} Unfortunately, complex biomechanical laboratories with costly and labor-intensive measurement tools are required to test individual athletes. These limitations restrict the potential to perform athlete risk assessments on a large scale, thereby limiting our ability to target athletes at higher injury risk with the appropriate intervention strategies. The defined clinical correlates to laboratory-measured knee biomechanics associated with increased risk of PFP yielded a highly sensitive model to predict increased KAM status. Combining these correlates in clinical screening algorithms that use measurements derived from a standard camcorder and a calibrated physician's balance scale may allow us to identify athletes at increased risk of PFP who may benefit from a targeted neuromuscular training intervention. These clinically feasible methods provide an indication of the relevance of identifying and ameliorating potential predisposing risk factors to prevent this syndrome that affects a large percentage of young female athletes. Implementing targeted trunk- and hip-strengthening and neuromuscular training at an earlier age for high-risk athletes may help to minimize lower extremity pathomechanics and therefore reduce the risk and occurrence of PFP before these athletes participate in high school sports.

REFERENCES

1. Fairbank JC, Pynsent PB, van Poortvliet JA, Phillips H. Mechanical factors in the incidence of knee pain in adolescents and young adults. *J Bone Joint Surg Br.* 1984;66(5):685–693.
2. Blond L, Hansen L. Patellofemoral pain syndrome in athletes: a 5.7-year retrospective follow-up study of 250 athletes. *Acta Orthop Belg.* 1998;64(4):393–400.
3. Fulkerson JP, Arendt EA. Anterior knee pain in females. *Clin Orthop Relat Res.* 2000;372:69–73.
4. Myer GD, Ford KR, Barber Foss KD, et al. The incidence and potential pathomechanics of patellofemoral pain in female athletes. *Clin Biomech (Bristol, Avon).* 2010;25(7):700–707.
5. Myer GD, Ford KR, Hewett TE. Quantification of knee load associated with increased risk for specific knee injury incidence [abstract]. *J Athl Train.* 2011;46(3):S-30.
6. Myer GD, Ford KR, Khoury J, Succop P, Hewett TE. Biomechanics laboratory-based prediction algorithm to identify female athletes with high knee loads that increase risk of ACL injury. *Br J Sports Med.* 2011;45(4):245–252.
7. Ford KR, Myer GD, Hewett TE. Reliability of landing 3D motion analysis: implications for longitudinal analyses. *Med Sci Sports Exerc.* 2007;39(11):2021–2028.
8. Quatman CE, Ford KR, Myer GD, Paterno MV, Hewett TE. The effects of gender and pubertal status on generalized joint laxity in young athletes. *J Sci Med Sport.* 2008;11(3):257–263.
9. Myer GD, Ford KR, Paterno MV, Nick TG, Hewett TE. The effects of generalized joint laxity on risk of anterior cruciate ligament injury in young female athletes. *Am J Sports Med.* 2008;36(6):1073–1080.
10. Myer GD, Ford KR, Barber Foss KD, Liu C, Nick TG, Hewett TE. The relationship of hamstrings and quadriceps strength to anterior cruciate ligament injury in female athletes. *Clin J Sport Med.* 2009;19(1):3–8.
11. Brent J, Myer GD, Ford KR, Paterno M, Hewett T. The effect of sex and age on isokinetic hip-abduction torques. *J Sport Rehabil.* 2013;22(1):41–46.
12. Myer GD, Brent JL, Ford KR, Hewett TE. A pilot study to determine the effect of trunk and hip focused neuromuscular training on hip and knee isokinetic strength. *Br J Sports Med.* 2008;42(7):614–619.
13. de Leva P. Joint center longitudinal positions computed from a selected subset of Chandler's data. *J Biomech.* 1996;29(9):1231–1233.
14. Harrell FE. *Regression Modeling Strategies with Application to Linear Models, Logistic Regression, and Survival Analysis.* New York, NY: Springer Publishing Co; 2001.

15. Hootman JM, Dick R, Agel J. Epidemiology of collegiate injuries for 15 sports: summary and recommendations for injury prevention initiatives. *J Athl Train.* 2007;42(2):311–319.
16. Willson JD, Binder-Macleod S, Davis IS. Lower extremity jumping mechanics of female athletes with and without patellofemoral pain before and after exertion. *Am J Sports Med.* 2008;36(8):1587–1596.
17. MacIntyre NJ, Hill NA, Fellows RA, Ellis RE, Wilson DR. Patellofemoral joint kinematics in individuals with and without patellofemoral pain syndrome. *J Bone Joint Surg Am.* 2006;88(12):2596–2605.
18. Hewett TE, Myer GD, Ford KR, Slauterbeck JR. Preparticipation physical exam using a box drop vertical jump test in young athletes: the effects of puberty and sex. *Clin J Sport Med.* 2006;16(4):298–304.
19. Hewett TE, Myer GD, Ford KR. Decrease in neuromuscular control about the knee with maturation in female athletes. *J Bone Joint Surg Am.* 2004;86-A(8):1601–1608.
20. MacKinnon CD, Winter DA. Control of whole body balance in the frontal plane during human walking. *J Biomech.* 1993;26(6):633–644.
21. Hewett TE, Myer GD. The mechanistic connection between the trunk, hip, knee, and anterior cruciate ligament injury. *Exerc Sport Sci Rev.* 2011;39(4):161–166.
22. Hewett TE, Myer GD, Ford KR. Reducing knee and anterior cruciate ligament injuries among female athletes: a systematic review of neuromuscular training interventions. *J Knee Surg.* 2005;18(1):82–88.
23. Hewett TE, Myer GD, Ford KR, et al. Biomechanical measures of neuromuscular control and valgus loading of the knee predict anterior cruciate ligament injury risk in female athletes: a prospective study. *Am J Sports Med.* 2005;33(4):492–501.
24. Ford KR, Myer GD, Hewett TE. Longitudinal effects of maturation on lower extremity joint stiffness in adolescent athletes. *Am J Sports Med.* 2010;38(9):1829–1837.
25. Ford KR, Shapiro R, Myer GD, Van Den Bogert AJ, Hewett TE. Longitudinal sex differences during landing in knee abduction in young athletes. *Med Sci Sports Exerc.* 2010;42(10):1923–1931.
26. Powers CM. The influence of altered lower-extremity kinematics on patellofemoral joint dysfunction: a theoretical perspective. *J Orthop Sports Phys Ther.* 2003;33(11):639–646.
27. Perry J. *Gait Analysis: Normal and Pathological Function.* Thorofare, NJ: Slack; 1992.
28. Winter DA. *Biomechanics and Motor Control of Human Movement.* 3rd ed. Hoboken, NJ: John Wiley & Sons; 2005.
29. Hewett TE, Ford KR, Myer GD, Wanstrath K, Schepers M. Gender differences in hip adduction motion and torque during a single-leg agility maneuver. *J Orthop Res.* 2006;24(3):416–421.
30. Boling MC, Padua DA, Marshall SW, Guskiewicz K, Pyne S, Beutler A. A prospective investigation of biomechanical risk factors for patellofemoral pain syndrome: the Joint Undertaking to Monitor and Prevent ACL Injury (JUMP-ACL) cohort. *Am J Sports Med.* 2009;37(11):2108–2116.
31. Besier TF, Lloyd DG, Cochrane JL, Ackland TR. External loading of the knee joint during running and cutting maneuvers. *Med Sci Sports Exerc.* 2001;33(7):1168–1175.
32. Li G, Rudy TW, Sakane M, Kanamori A, Ma CB, Woo SL. The importance of quadriceps and hamstring muscle loading on knee kinematics and in-situ forces in the ACL. *J Biomech.* 1999;32(4):395–400.
33. Myer GD, Ford KR, Khoury J, Succop P, Hewett TE. Development and validation of a clinic-based prediction tool to identify female athletes at high risk for anterior cruciate ligament injury. *Am J Sports Med.* 2010;38(10):2025–2033.
34. Palmieri-Smith RM, McLean SG, Ashton-Miller JA, Wojtyś EM. Association of quadriceps and hamstrings cocontraction patterns with knee joint loading. *J Athl Train.* 2009;44(3):256–263.
35. Myer GD, Ford KR, Hewett TE. The effects of gender on quadriceps muscle activation strategies during a maneuver that mimics a high ACL injury risk position. *J Electromyogr Kinesiol.* 2005;15(2):181–189.
36. Quatman CE, Kiapour A, Myer GD, et al. Cartilage pressure distributions provide a footprint to define female anterior cruciate ligament injury mechanisms. *Am J Sports Med.* 2011;39(8):1706–1713.
37. Beynon BD, Fleming BC. Anterior cruciate ligament strain in-vivo: a review of previous work. *J Biomech.* 1998;31(6):519–525.
38. Ford KR, Myer GD, Schmitt LC, Uhl TL, Hewett TE. Preferential quadriceps activation in female athletes with incremental increases in landing intensity. *J Appl Biomech.* 2011;27(3):215–222.
39. Barber Foss KD, Hornsby M, Edwards NM, Myer GD, Hewett TE. Is body composition associated with an increased risk of developing anterior knee pain in adolescent female athletes? *Phys Sportsmed.* 2012;40(1):13–19.
40. Lee SP, Powers C. Association between functional hip abductor strength and hip joint kinematics and kinetics during a dynamic unipedal drop landing task. Paper presented at: 35th Annual Meeting of the American Society of Biomechanics; August 10–13, 2011; Long Beach, CA.
41. Beynon B, Slauterbeck J, Padua D, et al. Update on ACL risk factors and prevention strategies in the female athlete. Paper presented at: 52nd Annual Meeting and Clinical Symposia of the National Athletic Trainers' Association; June 19–23, 2001; Los Angeles, CA.
42. Pataky TC, Zatsiorsky VM, Challis JH. A simple method to determine body segment masses in vivo: reliability, accuracy, and sensitivity analysis. *Clin Biomech (Bristol, Avon).* 2003;18(4):364–368.
43. Quatman CE, Ford KR, Myer GD, Hewett TE. Maturation leads to gender differences in landing force and vertical jump performance: a longitudinal study. *Am J Sports Med.* 2006;34(5):806–813.
44. Padua DA, Marshall SW, Beutler AI, et al. Predictors of knee valgus angle during a jump-landing task [abstract]. *Med Sci Sports Exerc.* 2005;37(5):S398.
45. Myer GD, Ford KR, Palumbo JP, Hewett TE. Neuromuscular training improves performance and lower-extremity biomechanics in female athletes. *J Strength Cond Res.* 2005;19(1):51–60.
46. LaBella CR, Huxford MR, Smith TL, Cartland J. Preseason neuromuscular exercise program reduces sports-related knee pain in female adolescent athletes. *Clin Pediatr (Phila).* 2009;48(3):327–330.
47. Myer GD, Ford KR, Brent JL, Hewett TE. Differential neuromuscular training effects on ACL injury risk factors in “high-risk” versus “low-risk” athletes. *BMC Musculoskelet Disord.* 2007;8:39.
48. Witvrouw E, Lysens R, Bellemans J, et al. Intrinsic risk factors for the development of anterior knee pain in an athletic population: a two-year prospective study. *Am J Sports Med.* 2000;28(4):480–489.

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