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Chronic Traumatic Encephalopathy: The Horse Is Still Chasing the Cart

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Chronic traumatic encephalopathy (CTE) occupies a unique and expanding position in both the scientific and lay communities concerned with sport participation at all levels. The popularity of sports with a high risk of repetitive head impacts, such as American football, drives intense and passionate media involvement compared to other areas of public interest in which medical research plays a role. The resulting benefits of rapid, widespread reporting and increased public awareness are counterbalanced by misinformation and an oversimplification of an increasingly complex topic.[2] To date, there are approximately 300 confirmed CTE cases described in the literature, the vast majority of which involve former professional athletes.

Though concussion and CTE are often considered in the same conversation, they represent starkly different conditions that are connected in a poorly understood way. In athletics, sport-related concussion is a transient, functional neurological disturbance wherein symptoms typically resolve in a matter of weeks, often within 7 to 10 days in adults. Chronic traumatic encephalopathy is a neuropathologically defined, potential long-term outcome observed in a subset of individuals with a history of extensive exposure to repetitive head impacts, often both concussive and "subconcussive" in nature.[4] Traumatic encephalopathy syndrome (TES) refers to the proposed clinical syndrome associated with underlying CTE pathology, but possibly other proteinopathies as well.[9] Hereafter, CTE will be used when referring to neuropathological signs and TES will be used when describing clinical symptomatology.

In this Viewpoint, we will describe the current understanding of CTE pathology and proposed criteria for characterizing TES. We want to emphasize that CTE and TES are not the same entity, at least according to our current understanding. We additionally highlight those at greatest risk for CTE and the significant challenges to symptom attribution specific to these populations.

Chronic Traumatic Encephalopathy

In 2015, a working group of neuropathologists proposed provisional diagnostic criteria for the pathological features of CTE following a consensus meeting.[6] The defining pathological signature of CTE was identified as abnormal deposition of hyperphosphorylated tau protein (proteins within the central nervous system that stabilize microtubules) surrounding blood vessels predominantly at the depths of cortical sulci. There was general, but not universal, agreement among the contributing neuropathologists that this tau deposition pattern was unique to CTE and that it differentiated the disorder from tauopathies such as Alzheimer's disease and other neurodegenerative conditions.

Chronic traumatic encephalopathy is almost always referred to as a progressive, neurodegenerative tauopathy.[6,7] Importantly, more work is needed to clarify the nature of disease progression, to refine neuropathological staging, to characterize the role of other proteins (eg, amyloid, TDP-43, alpha-synuclein, and others), and to determine whether some cases actually remain stable over time (ie, nonprogressive).[10] This requires longitudinal research and validation of biomarkers that can track disease severity, as opposed to the purely cross-sectional nature of current data.

Tau is considered the primary culprit in CTE. However, as is the case with

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many other neurodegenerative conditions, multiple pathological proteins are often observed. Beta-amyloid, a protein linked to Alzheimer's disease, has been described as a relatively common finding in CTE cases and seems to positively correlate with age at death.[11] Unsurprisingly, and once again similar to other diseases, many diagnosed with CTE also meet pathological diagnostic criteria for other neurodegenerative conditions.[7,10]

Traumatic Encephalopathy Syndrome

Translating underlying neuropathology to clinical symptomatology is complicated by many coexisting neurobiological and psychosocial factors that influence an individual's cognition, mood, and behavior. Accordingly, it is critical to conceptualize CTE and TES as associated, but far from perfectly correlated, constructs. It is probably unreasonable to expect that a single typical and reproducible TES profile will emerge that sensitively and specifically predicts CTE. The currently available data include fewer than 300 individuals with autopsy-confirmed CTE, whose clinical symptoms were almost exclusively derived from subjective reports of family members rather than from direct evaluations of the players themselves. That being said, the proposed provisional criteria for TES represent an important step in the ultimate development of clinically oriented means of diagnosing CTE with any certainty in living individuals.

The TES criteria proposed by Montenigro and colleagues,[9] though not yet validated for clinical application, arguably represent the most methodologically rigorous attempt to date, and emphasize sensitivity over specificity. In brief, the Montenigro criteria[9] require (1) a history of "multiple head impacts"; (2) objective cognitive decline in at least 1 of the categories of (*a*) episodic memory, executive function, or attention, as determined by neuropsychological evaluation, (*b*) significant behavioral changes, or (*c*) overly depressed mood; (3) symptoms lasting more than 1 year; (4) at least 2 supportive features, such as new-onset impulsivity, anxiety, or aggressiveness; and (5) the determination that the symptoms are not better explained primarily by another neurological condition, though comorbid diagnoses may be common.

After determining that TES criteria are met, a specific TES subtype may be assigned based on the clinical symptom domains present: behavioral/mood, cognitive, or mixed variant. Some individuals could present with parkinsonian motor features. Motor symptoms were a primary feature of the more classically described dementia pugilistica, or "punch-drunk syndrome," but are rarely seen in modern cases. Recent studies disproportionately include American football players rather than boxers, who comprised the bulk of earlier samples. If CTE and dementia pugilistica indeed share pathological underpinnings, then the disparate clinical profiles suggest that there may be variance in the anatomical regions most affected by the pathology, potentially due to mechanical differences in the impacts sustained by American football players versus boxers.

An important point often confused in media portrayals and misinterpreted by the general public is the belief that having CTE is synonymous with having dementia. Dementia and TES are clinically defined conditions describing the symptoms that patients display when they present for medical evaluation, while CTE is purely a neuropathological description. Dementia differs from TES in that having dementia requires that an individual be significantly impaired in day-to-day functioning, such that he or she is unable to independently perform activities like personal care (eg, dressing and bathing oneself) and other instrumental activities, such as managing finances, cooking, driving, etc. The Montenigro criteria[9] contain a specifier for TES dementia if loss of independent functioning is present, but it is otherwise inappropriate to indiscriminately apply the term *dementia* to all those suspected of having CTE or TES.

Available data do not yet support the ability to diagnose CTE with any degree of meaningful certainty while individuals are alive. Chronic traumatic encephalopathy, like other neurodegenerative conditions, can only be definitively diagnosed at autopsy. Where CTE lags behind conditions like Alzheimer's disease is the ability for clinicians to utilize clinical symptomatology and biomarkers to inform a CTE diagnosis with acceptable sensitivity or specificity. As mentioned, this is to be expected given the relative infancy of this research.

At-Risk Populations

The strongest known risk factor for CTE is exposure to repetitive head trauma. The necessary and sufficient extent of exposure is unknown, as are individual-level risk factors (for instance, why an equivalent exposure to head trauma does not pose the same risk to different individuals). One of the biggest hurdles for the advancement of the science of CTE risk factors is poor understanding of CTE's prevalence and incidence. To fill this knowledge gap, all the combinations of (1) those with and without exposure to repetitive head trauma, (2) those with and without CTE, and (3) those with and without TES must be studied.[2]

Repetitive head trauma has been identified as the strongest risk factor, primarily because the vast majority of individuals studied have had such exposure. The target population throughout the earlier part of the 20th century, though studied on a much smaller scale than are the at-risk populations of today, was boxers. Currently, American football players are under the microscope, as are, to a lesser but growing extent, soccer, hockey, and rugby players. Military personnel with frequent blast exposure may share a similar risk, though the fact that many in the military also played high-risk collision sports often presents difficulties with determining etiology. There are sporadic reports of individuals with CTE-like pathology, despite no known history of head impacts; however, the likelihood that

such cases may exhibit pathognomonic CTE largely remains unknown.

The specific roles played by concussion (diagnosed or not) and subconcussive head impacts in the development of CTE are somewhat controversial. The phrase *subconcussive impacts* refers to impacts common in collision sports, like American football, where forces are transmitted to the brain but no clinical symptoms result. Studies examining associations between brain trauma history and the extent of CTE pathology have evaluated both concussion history and subconcussive impacts, typically defined either by the cumulative years playing collision sports or using an estimate of the number of impacts sustained throughout the course of a career. Results are somewhat variable, but concussion history (ie, the number of self-reported diagnosed and undiagnosed concussions) does not seem to correlate well with disease severity. Conversely, there is stronger, though still inconsistent, evidence linking either years playing collision sports or estimated number of career head impacts to CTE severity.[7,8]

Taken together, collision-sport athletes and certain military personnel appear to be at greatest risk of developing CTE, though just how great the risk is remains unclear. These populations are patently different from their counterparts in the general civilian population. Concussion is certainly common among civilians and nonathletes, owing primarily to motor vehicle accidents or falls. However, single or even multiple isolated instances of concussion are not equivalent to the type of exposure to both concussive and subconcussive trauma seen in collision sports. This is important both clinically and when interpreting brain injury research that describes disparate populations.[1]

Challenges to Understanding Clinical Outcomes

Athletes who play collision sports, particularly retired American football players, more often present with characteristics and experiences that complicate the ability to determine the cause of cognitive, mood, and behavioral symptoms. Such factors include sleep problems, chronic pain, difficulties adjusting to postathlete lifestyles, substance use, and others.[2,3] Briefly, persistent sleep disturbances may increase the risk for CTE-related neuropathology and may produce symptoms similar to those described in TES.[12] Athletes may have significant mood and behavioral problems when they retire from sport due to shifts in financial status, loss of personal identity, absence of an outlet for aggression, etc. How these problems interact with brain trauma history is unclear, but they importantly represent potential targets of treatment designed to mitigate symptom burden and improve quality of life.

The influences of both medical comorbidities and sociodemographic factors highlight the critical importance of comprehensive clinical examinations, including extensive patient histories and exploring multiple domains of cognition, mood, and behavior. Additionally, elucidating the timing of symptom onset and progression of symptoms over time is essential for differential diagnosis. For example, mood or behavior symptoms that present immediately after retirement may represent a different process than do similar symptoms presenting years later, in the absence of any life stressors. Symptoms that dissipate significantly with treatments such as sleep therapy, psychotherapy, and/or medication might suggest a nonneurodegenerative source. Practically no data exist on the extent to which TES-related symptoms are treatable. The disproportionate prevalence of these complicating factors in athlete populations once again highlights the uniqueness of this area of clinical and research endeavors.

Appropriate Concern Versus Unfounded Fears

It is no longer possible to ignore the potential risks of extended involvement in sports that involve repetitive head impacts, but it is important for athletes to know that collision-sport participation, especially at youth, high school, and collegiate levels, far from guarantees development of CTE/TES. The physical and mental benefits of sport participation are undeniable and should not be overlooked in these discussions. The ultimate determination of risks versus benefits of prolonged collision-sport participation is athlete, parent, and family specific. This determination should be made based on the best available scientific, objective data, and not on news headlines. Many athletes are acutely aware of CTE because of the media attention this problem has received. The messages portrayed, however, rarely reflect the reality that there is much still to be learned about CTE and TES. The perceived link between CTE and suicide is particularly concerning for many athletes and their families, despite a lack of empirical evidence to support this association (for review, see Iverson[5]).

It is very likely that ongoing research will show what is already known to be true in other diseases like Alzheimer's disease: the presence of CTE pathology, especially milder cases, does not necessarily mean that the individual will go on to develop TES symptoms or dementia. There are already a handful of cases in the literature of individuals with CTE pathology discovered at autopsy who were asymptomatic during life.[7] As we learn more about those who are able to carry certain levels of CTE pathological burden without showing symptoms, proactive interventions may be developed to help preserve cognitive, mood, and behavioral functioning.

As discussed, clinicians should not yet be diagnosing living individuals with CTE due to the lack of a validated clinical profile or CTE biomarkers. Diagnosing someone with CTE carries a great risk of introducing iatrogenic fear and anxiety, as well as an unwillingness to seek treatment, given that many perceive CTE as tantamount to dementia and an impending death sentence. The

most helpful message is one of encouragement to disclose concerns and seek professional treatment for troubling symptoms. Referral to medical providers such as neuropsychologists and neurologists experienced with these populations will help clarify the source and severity of symptoms, and will provide the athlete with the best chance for good outcomes and preserved or improved quality of life.

Key Points

- Chronic traumatic encephalopathy is a neuropathological diagnosis made without respect to clinical symptoms, while TES is a clinical syndrome describing symptoms without respect to underlying neuropathology.
- The similarities of CTE to other neurodegenerative conditions like Alzheimer's disease are that (1) CTE can only be definitively diagnosed during autopsy, (2) comorbid pathology is commonplace, and (3) presence of CTE pathology does not guarantee presence or later development of clinical symptoms like TES.
- The greatest known risk factor for CTE is exposure to repetitive head impacts, usually both concussive and subconcussive. However, CTE prevalence, incidence, and other risk factors are currently unknown.
- Due to a lack of validated diagnostic criteria or biomarkers, CTE should not yet be diagnosed by clinicians. Symptoms associated with CTE often have multiple sources, many of which are treatable or modifiable, and the factors that complicate interpretation of CTE/TES are disproportionately prevalent in athlete populations.
- Current and former athletes should be encouraged to report and discuss concerns about CTE or symptoms they may be experiencing so they can be referred appropriately. This will help clarify the source and severity of symptoms, inform treatment options, and, in active players, contribute to the decision about whether to keep playing their sport.

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Risk Factors for Groin Injury and Groin Symptoms in Elite-Level Soccer Players: A Cohort Study in the Dutch Professional Leagues

BACKGROUND: Groin injury and groin symptoms are common in soccer players. The relationship of groin injury and groin symptoms to reduced hip range of motion (ROM) and previous injury is unclear.

OBJECTIVES: To conduct a retrospective assessment of associations between previous injury and preseason hip ROM and preseason prevalence of severe groin symptoms, and to prospectively identify risk factors for within-season groin injury.

METHODS: During the period of 2015 to 2016, 190 players from 9 Dutch professional soccer clubs participated in this cohort study with prospective and retrospective elements. Univariate and multivariate logistic regressions were used to predict preseason severe groin symptoms, identified using the Copenhagen Hip and Groin Outcome Score, from a history of previous groin injury, general injury (minimum of 1 week in duration) in the previous season, and hip ROM. Cox regression was used to predict within-season groin injury.

RESULTS: Point prevalence of severe groin symptoms was 24% and within-season incidence of groin injury was 11%. Total, training, and match groin injury incidences were 0.5, 0.2, and 2.6 injuries per 1000 playing hours, respectively. A history of more than 1 previous groin injury was associated with current severe groin symptoms (odds ratio = 3.0; 95% confidence interval: 1.0, 8.3; $P = .038$). General injury sustained in the previous season (ankle, knee, thigh, shoulder; median, 9 weeks of time loss) was a risk factor for groin injury (hazard ratio = 5.1; 95% confidence interval: 1.8, 14.6; $P = .003$).

CONCLUSION: Severe injuries in the previous season to locations other than the groin increase the risk of groin injury the next season. A history of groin injury is associated with current severe groin symptoms. Preseason hip ROM does not identify players at risk for groin injury.

LEVEL OF EVIDENCE: Prevention, level 2b.
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Muscle injury is very common in professional soccer players and accounts for one third of all time-loss injury.[2,7,14] Injury to 4 major muscle groups of the lower extremity accounts for more than 90% of all muscle injuries.[6] Injury prevalence per muscle group is 25% for adductors, 42% for hamstrings, 19% for quadriceps, and 14% for calves.[14] Recurrence rates are highest for the adductors (29%) and hamstrings (30%), and overuse injuries (34%) are mostly adductor related in elite European soccer players.[14] A recent systematic review demonstrated that adductor or groin injury[41] is particularly problematic due to high incidence and risk of chronicity and recurrence.[43]

Each season, up to 19% of all professional soccer players sustain a time-loss groin injury,[11,39] with an average time loss between 15 and 20 days,[6,8,13,27,39,42] and more than 28 days for recurrent groin injury.[7] Groin injury incidence rates vary between European countries[40]; for example, total, training, and match groin injury incidences were 0.7, 0.4, and 2.9 injuries per 1000 playing hours for a Dutch cohort,[27] while they were 1.3, 1.0, and 3.7 injuries per 1000 playing hours for a Swedish cohort.[12] Time-loss injury data underestimate the full extent of the injury burden, as groin symptoms

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are highly prevalent in professional soccer,[29,32,34,39] and many players continue to train and play with pain.[15,29,34]

Previous groin injury is a known risk factor for a future groin injury,[43] yet it is rarely considered that a previous injury may increase the risk of injury to a different location.[10] A recent systematic review demonstrated that there is an injury relationship between various lower extremity muscles.[35] Another study showed a relationship between previous knee injury and subsequent groin injury.[37] Although subsequent injuries are common, the relationship with previous injuries to different locations is not well understood.[10] Another recent systematic review identified lower total hip range of motion (ROM) of both hips as the most consistent factor related to the development of groin injury in athletes, whereas reduced hip internal rotation, abduction, and extension ROM were not related.[28] However, the literature is inconsistent on terms and definitions used for groin pain, injury, and hip ROM measurements.[28,38] In previous studies, athletes with current groin pain showed reduced hip internal rotation[21] and reduced hip ROM during the backswing of the soccer kick.[31]

Accordingly, the first aim of this study was to examine point prevalence of severe preseason groin symptoms and their association with previous injuries and hip ROM at baseline. The second objective was to examine the within-season groin injury incidence rate and risk factors for prospective groin injury during the 2015-2016 season.

METHODS

Design

This was a cohort study with prospective and retrospective elements. The protocol for the study, consistent with the requirements of the Declaration of Helsinki, was approved by the Medical Ethics Examination Committee (Amsterdam Medical Center) under number W15_086#15.0100. All participants gave their written informed consent.

Participants

Sixteen Dutch professional soccer clubs were invited to participate in the GRoin Injury Prevention study. To be included, players had to be 18 years of age or older, able to follow Dutch or English instructions, and capable of undergoing hip ROM testing.

Data were collected at baseline (preseason, June-July 2015) and within season (August 2015-May 2016) at the clubs' facilities (**FIGURE 1**). A standardized questionnaire was used to collect data on age (years), height (centimeters), weight (kilograms), body mass index (kilograms per square meter), and leg dominance (right/left, defined as the preferred kicking leg) at baseline.

Retrospective Injury Registration

A history of previous groin injury was recorded and categorized as (1) no history of injury; (2) yes, 1 injury; or (3) yes, more than 1 injury. General injury in the previous season was defined as time loss due to injury to any body region in the 2014-2015 season. Injury body location and time-loss duration, defined from the day of injury to the day being able to play a match,[1] were also recorded. Data for players with moderate (8-28 days of time loss) and severe (greater than 28 days of time loss) general injury[11] were recorded using dichotomous outcome options (yes/no), whereas history of minor general injury (1-7 days)[11] was not recorded.

Baseline Hip ROM

The test procedures to measure hip ROM were consistent with those used in previous studies.[22,31] Six physical therapists with 5 to 31 years of clinical experience performed 9 hours of training over 3 sessions to enhance uniformity of the testing procedures. Both hips were assessed using the following order: hip internal rotation, external rotation, abduction, and adduction. Range of motion of hip rotation was measured using a universal goniometer in increments of 1° (Baseline evaluation instruments; Fabrication Enterprises, Inc, White Plains, NY). The players were positioned in supine, with their hips and knees flexed to 90°.[20,23] For hip abduction and adduction ROM, the players were in a sidelying position, with the trunk contralaterally rotated and the nontested leg in 90° of hip and knee flexion using a digital inclinometer (Baseline evaluation instruments).[22]

All ROM measurements were performed twice and values were averaged. Assessors were blinded to leg dominance, injury history, and the presence or symptoms of injury. One assessor performed the test while the other recorded ROM

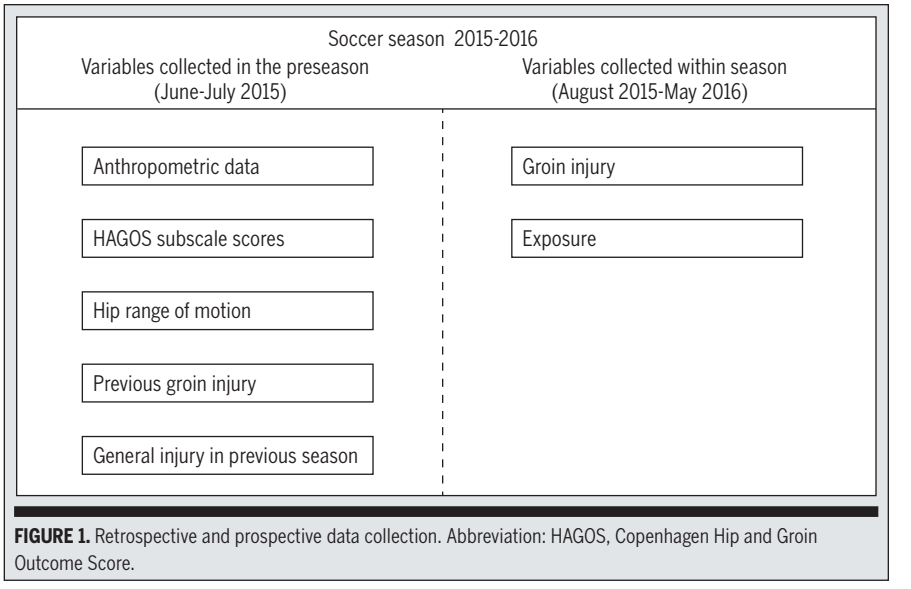

FIGURE 1. Retrospective and prospective data collection. Abbreviation: HAGOS, Copenhagen Hip and Groin Outcome Score.

values. These values were used to calculate ROM of several combinations of hip positions: total internal rotation, external rotation, adduction, and abduction of both hips; total internal and external rotation per hip and for both hips; total adduction and abduction per hip and for both hips; the difference between internal rotation, external rotation, adduction, and abduction of both hips; the difference between internal and external rotation per hip and between both hips; and the difference between adduction and abduction per hip and between both hips.

Intrarater and interrater reliability and precision of hip ROM measurement were determined prior to the study using 2 randomly selected assessors who tested 20 hips from 10 collegiate students on 2 occasions on the same day.[25,36]

Baseline Groin Symptoms

The Copenhagen Hip and Groin Outcome Score (HAGOS), a patient-reported outcome measure to assess levels of perceived hip and groin problems in young and active individuals,[33] was used to quantify groin symptoms at baseline. The HAGOS contains 37 items grouped into 6 separate subscales: pain, symptoms, physical function in daily living, physical function in sport and recreation, participation in physical activities, and hip and groin–related quality of life. Subscale scores range from 0 to 100, where 0 indicates extreme groin symptoms and problems and 100 indicates no symptoms or problems.[33] The HAGOS is available in the Dutch language[30] and is valid, reliable (reported intraclass correlation coefficients [ICCs; test-retest] ranging from 0.83 to 0.87), and internally consistent (Cronbach α = .81-.92). The smallest detectable change ranged from 17.7 to 32.0 points at the individual level and from 1.1 to 2.7 points at the group level.[29] These values are comparable to those of the original Danish version of the HAGOS.[33] Players proficient in Dutch completed the HAGOS online (Questback digital platform; Questback, Oslo, Norway), while the other players completed an officially translated paper version in English.

Based on the HAGOS scores at baseline, players who scored in the lower quartile (interquartile range [IQR], 0%-25%) for 2 or more subscales were allocated to the severe groin symptoms group. Players who scored in the upper quartile (IQR, 75%-100%) for 2 or more subscales formed the minor groin symptoms group. Players who scored in the upper or lower quartile for none or 1 subscale, with the rest of the subscales being in the middle 2 quartiles (IQR, 25%-75%), were referred to as "all others."[29] Subscale scores were calculated for the severe and minor groin symptoms groups, and both were included in retrospective analysis. The HAGOS scores from "all others" were not used.

Prospective Injury Registration

The club medical staff received instruction for within-season recording of time loss due to groin injuries according to the consensus statement on injury reporting in soccer.[11] A player was considered injured when any symptom of the front of the hip or the anteromedial part of the thigh was sustained during a soccer training or match and resulted in the inability to participate fully in at least 1 training session or match.[11] Groin injury was diagnosed according to the Doha consensus statement[41] and was classified for severity into minor (1-7 days), moderate (8-28 days), or severe (greater than 28 days), based on time loss.[11]

Exposure

Training and match exposures from August 2015 to May 2016 were recorded separately. Training exposure was defined as all individual and team physical activities led by a technical staff member, including friendly matches. Club medical staff recorded individual training exposure in minutes, using a standardized Excel form, and reported the data to the research team on a weekly basis. Individual match exposure was registered in minutes by Gracenote (Nielsen Holdings, New York, NY) and was reported each month. Causes of time loss from training or match play were recorded as (1) groin injury, (2) other injury, and (3) other reasons.

Statistical Analysis

Statistical analyses were performed using SPSS Version 23 (IBM Corporation, Armonk, NY). The Shapiro-Wilk test was used to confirm the normality of distribution of continuous variables. Normally distributed data are presented as mean ± SD and nonnormal data as median and IQR (25%-75%). Descriptive analyses were conducted for variables for the total group of players. For ROM measurements, generalized estimating equations were used to model the interdependency between dominant and nondominant hips.[30]

Hip ROM intrarater and interrater reliability ($ICC_{2,2}$) and minimal detectable change (MDC) at the 95% confidence interval (CI) were calculated. Training and match within-season exposures were calculated in hours for all players individually. Total, training, and match groin injury incidence rates were calculated per 1000 hours of exposure, with 95% CIs, by dividing the total number of injuries by total exposure time. Previous general injury was recorded for locations and time-loss duration in numbers and percent of the total.

For the retrospective analysis, univariate and multivariate logistic regression models were used to calculate associations between the severe groin symptoms group as the dependent variable (recorded at baseline) and player characteristics (age, height, weight, body mass index, leg dominance, previous groin injury, general injury in the previous season, hip ROM) as independent variables. For the prospective analysis, a Cox regression model was used. This model takes exposure into account and censors for abbreviated length of follow-up due to other injury or other reasons. In this model, the time (hours of exposure) from the start of the follow-up period (August 2015) until the event (groin injury) or the end of follow-up (May 2016) was the main variable. Formerly mentioned covariates (including severe

groin symptoms) were used as independent variables to identify risk factors for prospective groin injury as the dependent variable. Odds ratios (ORs) (logistic regression) and hazard ratios (Cox regression) and their 95% CIs were used to compare risks for severe groin symptoms and groin injury, respectively.

Absolute risks for severe groin symptoms and groin injury were calculated and reported as the percentage of players with and without the risk factor. For players with a previous general injury, a Mann-Whitney *U* test was used to compare differences in time-loss duration between those who sustained a future groin injury and those who did not.

RESULTS

Participants

From the 16 invited Dutch professional soccer clubs (362 players), 11 teams (239 players) from the Premier League (Eredivisie, 9 teams) and First Division (Jupiler League, 2 teams) agreed to participate. From these, 2 teams (41 players) were lost to follow-up, as the club management did not approve the preseason physical testing. Eight players were excluded from analysis: 4 players were unable to perform ROM tests due to acute groin and low back injury, and another 4 did not complete the HAGOS due to language issues. The remaining 190 players were included for retrospective analysis (53%). Ten players with general injury in the previous season who were diagnosed with groin injury were excluded from the risk-factor analysis between previous general injury to locations other than the groin and prospective groin injury. Another 9 were excluded, as they had no exposure in anticipation of pending transfers. The remaining 171 players were included for prospective analysis (47%) (**FIGURE 2**). There were no missing data.

At baseline, 25 players (13%) reported more than 1 previous groin injury and 39 players (21%) reported 1 previous groin injury. There were 91 players (48%) who reported 108 general injuries in the previous season (2014-2015) (**TABLE 1**). Numbers and locations of previous general injury were: 31 knee/lower leg (29%), 29 ankle/foot (27%), 18 thigh (16%), 10 hip/groin (9%), 8 shoulder (7%), 5 spine (4%), 4 wrist (4%), and 3 head (3%). There were 28 moderate (26%) and 80 severe (74%) general injuries.

Hip ROM

For intrarater reliability, ICCs were 0.95 (95% CI: 0.95, 0.98) for internal rotation, 0.88 (95% CI: 0.68, 0.96) for external rotation, 0.79 (95% CI: 0.37, 0.93) for abduction, and 0.95 (95% CI: 0.81, 0.98) for adduction. The corresponding MDC_{95} values ranged from 6° to 8°. For interrater reliability, ICCs for the same 4 motions were 0.86 (95% CI: 0.64, 0.95), 0.77 (95% CI: 0.38, 0.92), 0.58 (95% CI: 0.35, 0.87), and 0.84 (95% CI: 0.52, 0.95), respectively. The corresponding MDC_{95} values ranged from 8° to 11°.

Baseline hip ROM values are provided in **TABLE 2**, and combinations of hip ROM measurements are provided in the **APPENDIX** (available at www.jospt.org).

Baseline Prevalence of Groin Symptoms

The preseason point prevalence for players with severe groin symptoms was 24% (46 players), and for those with minor groin symptoms was 60% (114 players). The HAGOS subscale scores are provided in **TABLE 3**.

Retrospective Associations With Severe Groin Symptoms

Univariate logistic regression analysis revealed that more than 1 previous groin injury was associated with baseline severe groin symptoms (OR = 3.7; 95% CI: 1.4, 9.9; P = .009). Multivariate logistic regression analysis with previous general injury and the difference in adduction between both hips as potential risk factors (P<.1 from univariate analysis) confirmed

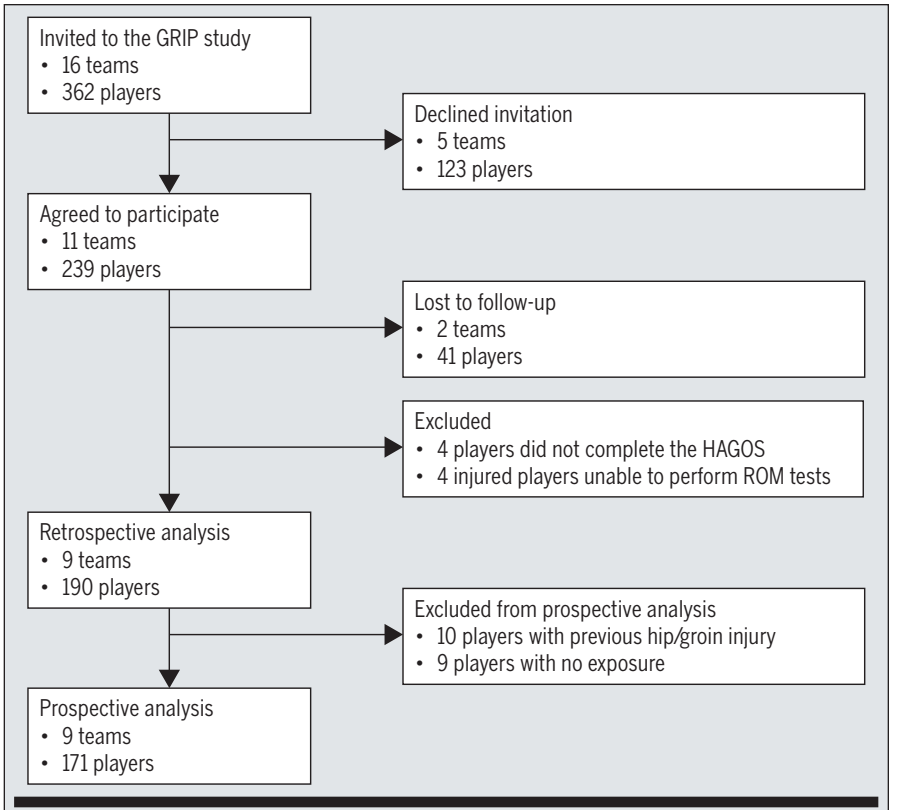

FIGURE 2. Flow chart of inclusion and exclusion of players in the study. Abbreviations: GRIP, GRoin Injury Prevention; HAGOS, Copenhagen Hip and Groin Outcome Score; ROM, range of motion.

TABLE 1 Baseline Characteristics for All Players (n = 190)

Characteristic	Value
Age, y*	23.3 ± 3.8
Height, cm*	182.4 ± 7.2
Weight, kg*	77.8 ± 7.3
BMI, kg/m^2*	23.4 ± 1.4
Leg dominance, n (%)	
Right	150 (79)
Left	40 (21)
Previous groin injury, n (%)	
No	126 (66)
Yes, 1 injury	39 (21)
Yes, >1 injury	25 (13)
General injury in previous season, n (%)†	
No injury or minor injury (<1 wk)	99 (52)
Yes, moderate or severe injury (>1 wk)	91 (48)
HAGOS subscale scores‡	
Pain	98 (93-100)
Symptoms	86 (79-93)
Function in daily living	100 (95-100)
Function in SR	97 (91-100)
Participation in PA	100 (88-100)
Hip- and/or groin-related QoL	90 (75-100)

Abbreviations: BMI, body mass index; HAGOS, Copenhagen Hip and Groin Outcome Score; PA, physical activity; QoL, quality of life; SR, sport and recreation.
**Values are mean ± SD.*
†Severity of general injury was classified as minor (1-7 days), moderate (8-28 days), or severe (greater than 28 days), based on time loss.
‡Values are median (interquartile range).

TABLE 2 Hip Range-of-Motion Values for All Players (n = 190)*

Movement/Side	Value
Internal rotation	
Dominant	19 ± 9
Nondominant	20 ± 10
External rotation	
Dominant	36 ± 8
Nondominant	36 ± 8
Abduction	
Dominant	37 ± 8
Nondominant	35 ± 9
Adduction	
Dominant	22 ± 6
Nondominant	22 ± 6

**Values are mean ± SD degrees.*

the association of more than 1 previous groin injury with severe groin symptoms (OR = 3.0; 95% CI: 1.0, 8.3; P = .038) (**TABLE 4**). Absolute risk findings showed that 44% of the players with more than 1 previous groin injury had severe groin symptoms during the preseason, compared to 15% of those with 1 previous groin injury and 23% of those with no previous groin injury.

Exposure

Within-season exposure time was 37 543 hours, consisting of 33 483 training hours and 4060 match hours. Each player spent an average ± SD of 198 ± 86 hours playing soccer, which included 176 ± 85 training hours and 21 ± 20 match hours during the 39-week competitive season.

Prospective Groin Injury

The within-season groin injury incidence rate was 0.5 injuries per 1000 playing hours (95% CI: 0.1, 0.5). The incidence rate was 0.2 (95% CI: 0.1, 0.4) injuries per 1000 training hours and 2.6 (95% CI: 1.5, 4.9) injuries per 1000 match hours.

A total of 24 groin injuries (11%) were reported in 18 players (9%). Diagnoses were adductor related (n = 16, 67%), iliopsoas related (n = 1, 4%), inguinal related (n = 1, 4%), pubic related (n = 5, 21%), and hip related (n = 1, 4%). Four players sustained 1 reinjury and 1 player sustained 2 reinjuries. There were 8 minor (33%), 10 moderate (42%), and 6 severe (25%) groin injuries, resulting in a median time loss of 13 days per player per season. Thus, an average squad of 21 ± 3 players can expect 2.7 groin injuries per season, resulting in 35 days lost to play.

Prospective Risk-Factor Analysis for Groin Injury

Univariate Cox regression analysis revealed that general injury in the previous season in regions other than the groin was a risk factor for groin injury (hazard ratio = 4.6; 95% CI: 1.6, 13.2; P = .004). Multivariate Cox regression analysis with weight and the difference in external rotation between both hips as potential

risk factors confirmed this risk factor relation (hazard ratio = 5.1; 95% CI: 1.8, 14.6; $P = .003$) (**TABLE 5**).

Absolute risk findings showed that 5% of the players without previous general injury sustained a future groin injury, compared to 15% of those with previous general injury. Post hoc analysis showed that the injury locations from the latter group were at the ankle (38%), knee (23%), shoulder (23%), and thigh (15%). These players (15%) had significant longer time loss in the previous season (median, 9 weeks; IQR, 8-22 weeks) than players with previous general injury who did not sustain a future groin injury (85%) (median, 6 weeks; IQR, 4-12 weeks; $P = .03$).

DISCUSSION

The main finding of this study is that elite-level soccer players with previous severe ankle, knee, thigh, and shoulder injury were at high risk to sustain a groin injury the next season. The within-season groin injury incidence was 11%, and an average team of 21 players can expect 3 time-loss groin injuries per season and 35 days of time loss. Severe groin symptoms were associated with a history of more than 1 previous groin injury. Preseason hip ROM testing did not identify individual players at risk of groin injury.

Baseline Prevalence of Severe Groin Symptoms

The high preseason point prevalence of severe groin symptoms (24%) in this study is consistent with the findings from Dutch (20%)[29] and Danish (36%)[34] cohorts, suggesting that players do not fully recover during the offseason. This prevalence of symptoms has been reported to increase during the competitive season to 29% (Norway),[15] 49% (Denmark),[34] and 55% (Sweden).[17] In this study, severe groin symptoms were defined as the lower quartile of HAGOS scores, as used in a previous study.[29] Median subscale scores for pain (86) and symptoms (75) in players with severe groin symptoms (**TABLE 3**) showed good agreement with Australian soccer players with current groin pain (pain, 88 and symptoms, 73).[5]

The high prevalence of severe groin symptoms reflects a serious problem in soccer across multiple cohorts and shows the relevance of assessing groin symptoms using more than only a time-loss groin injury definition.

TABLE 3 HAGOS Subscale Scores for Players With Severe (Lower Quartile) and Minor (Upper Quartile) Groin Symptoms*

HAGOS Subscale	Severe Groin Symptoms (n = 46)	Minor Groin Symptoms (n = 114)
Pain	86 (80-91)	100 (98-100)
Symptoms	75 (63-86)	89 (86-96)
Function in daily living	90 (85-95)	100 (100-100)
Function in SR	84 (74-91)	100 (97-100)
Participation in PA	88 (75-100)	100 (100-100)
Hip- and/or groin-related QoL	75 (60-85)	100 (90-100)

Abbreviations: HAGOS, Copenhagen Hip and Groin Outcome Score; PA, physical activity; QoL, quality of life; SR, sport and recreation.
**Values are median (25%-75% interquartile range).*

TABLE 4 Multivariate Logistic Regression Analysis (n = 190) for Associations Between Severe Groin Symptoms and Player Variables With $P<.10$ From the Univariate Model

Player Variable	Odds Ratio*	P Value
General injury in previous season	1.519 (0.700, 3.296)	.290
Difference in hip adduction range of motion between hips	1.161 (1.035, 1.303)	.011†
Greater than 1 previous groin injury	2.975 (1.061, 8.343)	.038†

**Values in parentheses are 95% confidence interval.*
†*P<.05.*

TABLE 5 Multivariate Cox Regression Analysis (n = 171) for Associations Between Prospective Groin Injury and Player Variables With $P<.10$ From the Univariate Model

Variable	Hazard Ratio*	P Value
Difference in hip external rotation range of motion hips	1.049 (0.944, 1.215)	.124
Weight	0.924 (0.869, 0.983)	.012†
General injury in previous season	5.070 (1.766, 14.562)	.003†

**Values in parentheses are 95% confidence interval.*
†*P<.05.*

Within-Season Groin Injury Incidence and Exposure

Groin injury incidences from this study (total, 0.5; training, 0.2; match, 2.6 injuries), and the prevalence of 11%, are comparable to a previous Dutch study in professional soccer players (total, 0.7; training, 0.4; match, 2.9; injury incidence and prevalence, 11%).[27] The small differences are reflective of seasonal exposure

findings from both studies (21/176 and 24/189[27] match-training player-hours, respectively).[16] These differences may be explained by decreased playing load of the Dutch national and competitive teams in recent years.

The reported exposure in a Swedish study[12] was about twice as high (53/340 match-training player-hours), which may explain higher groin injury incidence rates (total, 1.3; training, 1.0; match, 3.7)[12] for teams from Northern European countries.[40]

Retrospective Associations With Severe Groin Symptoms

The relationship between more than 1 previous groin injury and severe preseason groin symptoms is consistent with the outcomes of 2 previous studies.[29,34] Absolute risk findings showed that nearly half of the players with more than 1 previous groin injury had severe preseason groin symptoms. For clinicians, this means that successful and complete management of current groin injury may lower the burden of groin symptoms during the following preseason. Treatment should target the prevention of reinjury and not solely focus on lessening symptoms and enabling return to play.[35] From all combinations of hip measurements, only the difference in adduction between both hips was associated with severe groin symptoms (P = .011); however, this factor may be considered clinically nonrelevant (difference in adduction less than the MDC_{95}; OR = 1.2).

Risk Factors for Prospective Groin Injury

The main finding of this study is that players with general injury (duration of at least 1 week) in regions other than the groin had a greater risk (hazard ratio = 5.1; 95% CI: 1.8, 14.6) of sustaining a groin injury the next season compared to players without previous general injury. This important clinical finding adds to the recent insight that a previous injury may relate to a subsequent injury in a different location,[10,43] which is rarely considered.[35]

Some epidemiologic studies demonstrated relationships between injuries at different locations in the lower extremities[2,3,24,35] due to changes in running[24,37] and kicking[18,19,26] biomechanics that caused inadequate compensatory movement and motor control strategies.[14] The subsequent injury classification (SIC) model facilitates more accurate documentation of the within-player relationship.[3,9,10] It describes the first injury as the index injury and subsequent injuries as recurrent (identical in location and nature compared to the index injury), local (identical in location but different in nature), and new (different location).[9] It was demonstrated in Australian professional football that the majority of subsequent injuries were not related to the index injury (81%), yet 16% were local/recurrent and 3% were new index-related injuries.[10]

In the present study, 15% of the players with a previous general (index) injury sustained a new groin injury, suggesting a relationship between the index injury and subsequent injury. Additionally, these same individuals had significantly longer time loss in the previous season (median, 9 weeks) than the 85% of players with previous general injury who did not sustain a future groin injury (median, 6 weeks). Severe time loss results in unfit players due to a reduced training load.[44] It has been demonstrated that a reduced chronic workload followed by a high acute workload increases the risk of injury.[44] Given the 5-fold greater risk of groin injury throughout the next season, physical therapists should assess the extent of recovery from severe injuries in the previous season sustained to locations other than the groin.

Another important clinical finding of this study was that preseason hip ROM was not a useful screening tool for groin injury, even though a wide range of ROM variables were analyzed.[28] A strong relationship between reduced unilateral hip ROM and groin injury has never been demonstrated, and this study adds to that information.[3]

Study Strengths

A strength of this study was the use of 2 groin injury definitions—both time loss and symptoms—recorded using the HAGOS. Groin injuries were classified according to the Doha agreement meeting,[41] and a wide range of hip ROM combinations were studied.[28]

Study Limitations

The available sample for analysis limits the generalizability to the primary potential pool of 362 players, as 123 players declined the invitation, 41 were lost to follow-up, 8 were excluded for total analysis, and another 19 were excluded for prospective analysis, resulting in a sample of 190 players (52%) for retrospective and 171 players (47%) for prospective analysis. Although the sample size was good compared to many similar studies,[3,8,13] Cox regression analysis reduced power from 24 to 18 groin injuries. To detect moderate or strong associations, 20 to 50 cases are required.[4] Although used before,[28] severe groin symptoms, based on the lower quartile of HAGOS scores, have not yet been validated in the literature.

CONCLUSION

This study demonstrates that elite-level soccer players with severe injury to locations other than the groin in the previous season are at high risk for groin injury during the next season. Severe groin symptoms in the preseason are associated with a history of more than 1 previous groin injury. Physical therapists should assess the extent of recovery from severe injuries in the previous season sustained to locations other than the groin. Hip ROM testing during the preseason for screening purposes to identify soccer players at risk for groin injury does not seem valuable. ◉

KEY POINTS

FINDINGS: Elite-level soccer players with ankle, knee, thigh, and shoulder injury in the previous season and a median

time loss of 9 weeks are at 5.1 times greater risk of groin injury the following season compared to players who have less time loss or no previous injury. High preseason prevalence (24%) of severe groin symptoms is associated with a history of more than 1 groin injury. Preseason hip range of motion does not identify individual players at risk of within-season groin injury.

IMPLICATIONS: The findings of this study suggest that rehabilitation and training should ensure complete recovery of significant injuries sustained during the previous season. Persistent groin symptoms from groin injuries sustained in the previous season should be addressed during the offseason or early in the season.

CAUTION: The association of more than 1 previous groin injury with severe preseason groin symptoms should be interpreted with caution due to borderline coefficients. Due to the strong relationship between exposure and injuries, the low groin injury incidence in this study limits generalizability to soccer teams with higher or lower (amateur level) exposure.

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APPENDIX

HIP RANGE OF MOTION FOR COMBINATIONS OF HIP MEASUREMENTS FOR ALL PLAYERS (N = 190)*

Movement/Measurement	Value
Hip rotation	
Difference in internal rotation, both hips	5 ± 5
Difference in external rotation, both hips	5 ± 4
Total internal rotation, both hips	39 ± 18
Total external rotation, both hips	72 ± 15
Total internal and external rotation, dominant hip	55 ± 13
Total internal and external rotation, nondominant hip	55 ± 12
Total internal and external rotation, both hips	110 ± 24
Difference in total rotation, both hips	6 ± 6
Hip abduction and adduction	
Difference in abduction, both hips	5 ± 4
Difference in adduction, both hips	4 ± 3
Total abduction, both hips	72 ± 16
Total adduction, both hips	44 ± 12
Total abduction and adduction, dominant hip	59 ± 12
Total abduction and adduction, nondominant hip	57 ± 12
Total abduction and adduction, both hips	116 ± 23
Difference in abduction and adduction, both hips	6 ± 5

**Values are mean ± SD degrees.*

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Quadriceps Rate of Torque Development and Disability in Persons With Tibiofemoral Osteoarthritis

BACKGROUND: Declines in the ability to rapidly generate quadriceps muscle torque may underlie disability in individuals with tibiofemoral osteoarthritis.

OBJECTIVE: To determine whether quadriceps rate of torque development (RTD) predicts self-reported disability and physical performance outcomes in individuals with tibiofemoral osteoarthritis.

METHODS: This controlled laboratory, cross-sectional study assessed quadriceps strength and RTD in 76 individuals (55% female; mean ± SD age, 61.83 ± 7.11 years) with symptomatic and radiographic tibiofemoral osteoarthritis. Early (0-50 milliseconds), late (100-200 milliseconds), and overall peak RTDs were quantified in the symptomatic (involved) and contralateral limbs and used to calculate bilateral average values. Disability was assessed using the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) function subscale and 3 physical performance tests, including the (1) 20-m fast-paced walk, (2) 30-second chair stand, and (3) timed stair climb. Separate univariate regression models were used to determine the unique associations among measures of quadriceps RTD, WOMAC function score, and physical performance outcomes after accounting for quadriceps strength (change in R^2).

RESULTS: Greater involved-side late RTD and greater bilateral average early RTD were associated with faster walking (change in R^2 = 0.05, P = .013 and change in R^2 = 0.05, P = .043, respectively). Greater bilateral average late RTD was associated with faster walking (change in R^2 = 0.20, P<.001) and faster stair climb (change in R^2 = 0.11, P = .001). No quadriceps RTD variable was significantly associated with WOMAC function score (change in R^2 range, <0.01-0.017).

CONCLUSION: Involved-limb quadriceps RTD was weakly associated with physical performance outcomes, but not self-reported disability, in individuals with tibiofemoral osteoarthritis. Bilateral average quadriceps RTD was moderately associated with walking speed.

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KEY WORDS: *chair stand, self-reported function, stair climb, strength, walking speed*

Osteoarthritis (OA) is the most prevalent chronic medical condition worldwide,[5] and knee OA results in more years lived with disability compared to any other musculoskeletal condition.[23] A variety of factors may increase disability in individuals with knee OA, including pain,[16] psychosocial impairments,[35] and declines in lower extremity muscle function.[4,32] Normal quadriceps function is important for generating the lower extremity joint actions necessary for locomotion, joint stability, and load attenuation during such functional tasks as walking and stair climbing.[4] Quadriceps weakness is a common clinical impairment exhibited by individuals with knee OA[20,34] and may contribute to increased disability and radiographic disease progression.[28,32] Therefore, quadriceps strength is commonly targeted to decrease disability in patients with knee OA.[4]

Disability associated with knee OA is quantified using both self-reported measures (ie, Western Ontario and McMaster Universities Osteoarthritis Index [WOMAC] function subscale)[29] and quantitative physical performance assessments (ie, chair-stand test, timed stair climb, fast-paced walk).[9,41] Maintaining adequate quadriceps strength is important for preventing disability in individuals with knee OA.[26,32] However, many tasks of daily living (eg, ascending stairs) also require the rapid generation of quadriceps torque in addition to adequate quadriceps strength.[36] The ability to rapidly generate quadriceps torque is more predictive of physical performance compared to muscle strength in mobility-

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impaired older adults.[12,30] Additionally, rapid torque production typically deteriorates more rapidly with age compared to strength.[1] While the ability to quickly generate quadriceps torque has been assessed in a variety of patient populations,[6,41] the relationship between quadriceps rate of torque development (RTD) and disability in individuals with knee OA has yet to be determined.

Several factors regulate RTD, including neural activation,[7] as well as muscle fiber type and contractile properties of the muscle.[15] In addition to generation of peak RTD, determining quadriceps RTD at both early (ie, 0-50 milliseconds) and late (ie, 100-200 milliseconds) intervals following torque onset is important to understanding the unique physiological mechanisms that contribute to the ability to rapidly generate torque.[7,14,15]

Individuals with knee OA commonly demonstrate deficits in neuromuscular activation in addition to quadriceps weakness.[21] Deficits in neuromuscular activation may negatively influence early RTD intervals (ie, 0-50 milliseconds), thereby limiting physical function and increasing disability. Conversely, alterations in muscle quality (ie, ultrasound-measured echogenicity) or muscle architecture (ie, fascicle length and pennation angle) may influence late RTD intervals (ie, 100-200 milliseconds).[14] Moreover, declines in muscle function occur in both the involved and contralateral limbs in individuals with tibiofemoral OA.[8,13,34] Successful completion of a variety of activities of daily living (ie, fast-paced walking, rising from sitting, and stair negotiation) requires sufficient bilateral contributions from the quadriceps. Therefore, declines in bilateral quadriceps function may contribute to increased disability, regardless of whether tibiofemoral OA is present in one or both limbs.

The purpose of this study was to determine whether early ($RTD_{0\text{-}50\ ms}$), late ($RTD_{100\text{-}200\ ms}$), and peak (RTD_{Peak}) quadriceps RTDs predict disability, quantified as both self-reported disability and physical performance, in individuals with tibiofemoral OA, after accounting for quadriceps strength. Quadriceps RTD variables were calculated in the involved limb as well as the contralateral limb to determine the association between bilateral quadriceps RTD and disability. The authors hypothesized that greater quadriceps RTD would be associated with less self-reported disability and better physical performance during functional tasks. It is important to determine the association between quadriceps RTD and disability to explore the potential of quadriceps RTD as a therapeutic target for improving physical function in individuals with knee OA.

METHODS

This controlled laboratory, cross-sectional study used data obtained from participants enrolled in a larger randomized controlled trial (NCT02634814), and the authors have reported the present study per the Strengthening the Reporting of Observational Studies in Epidemiology checklist.[39] Main outcome measures were assessed during a single testing session in the following order: (1) self-reported disability, (2) quadriceps strength and RTD assessed during maximal voluntary isometric contraction (MVIC), and (3) physical performance. Quadriceps strength and RTD were assessed bilaterally and collected simultaneously during a single assessment.

First, the researchers assessed the most symptomatic knee (involved knee), reported by the participant as causing the most difficulty during daily activities. Physical performance assessments were consistent with the Osteoarthritis Research Society International recommendations,[9] and were completed in the following order: (1) 20-meter walk, (2) 30-second chair-stand test, and (3) stair climb. Physical performance outcome measures were always performed in the same order to minimize the potential influence of fatigue that could be caused if stair climbing was performed first. The Institutional Review Board at the University of North Carolina at Chapel Hill approved all methods, and all participants provided written consent prior to their involvement in the study.

Participants

Participants were primarily recruited through referrals from participating physicians within the Department of Orthopaedics at the University of North Carolina at Chapel Hill. Participants were between 40 and 75 years of age and exhibited symptomatic tibiofemoral OA in at least one limb, defined as (1) radiographic evidence of tibiofemoral OA in the medial or lateral compartment (grades 2 to 4 on the Kellgren-Lawrence [K-L] scale)[18] and (2) normalized WOMAC function subscale score greater than 31.[29] An experienced, fellowship-trained musculoskeletal radiologist determined all tibiofemoral K-L grades.

All participants demonstrated voluntary neuromuscular activation deficits, quantified as a central activation ratio less than 90%,[29] as this was an inclusion criterion for the larger clinical trial. The researchers excluded individuals with (1) a cardiovascular condition restricting exercise, (2) a pacemaker, (3) a neurodegenerative condition, (4) rheumatoid arthritis, (5) cancer, (6) neural sensory dysfunction over the knee, and (7) body mass index (BMI) greater than 35 kg/m^2. Also excluded were participants with history of a corticosteroid or hyaluronic acid injection within the previous 4 weeks, lower extremity orthopaedic surgery in the past year, a traumatic knee injury in the past 6 months, total knee or hip arthroplasty in either limb, or a diagnosed, nonreconstructed knee ligament tear. The larger randomized controlled trial evaluated gait biomechanics; therefore, this study had the information necessary to exclude individuals needing an assistive device during walking and pregnant women.

A previous study demonstrated that quadriceps RTD_{Peak} uniquely explained

7% to 15% of the variance in physical function in individuals with total knee arthroplasty, after accounting for quadriceps strength.[41] The authors chose to power this study to detect a change in R^2 of 7%, which was the smallest change in R^2 previously reported.[41] Therefore, the authors estimated they would need at least 71 participants to detect a change in R^2 of 0.067, with 80% power and an alpha level of .05, using 2 predictor variables (G*Power Version 3.1; Heinrich-Heine Universität, Düsseldorf, Germany).[11] The researchers recruited 5 additional participants (n = 76) to maintain statistical power in the event that outliers were removed from the final data set.

Quadriceps Strength and RTD

Isometric quadriceps strength and RTD were collected using a calibrated isokinetic dynamometer (HUMAC NORM; Computer Sports Medicine, Inc, Stoughton, MA). Participants were seated with hips and knees flexed to 85° and 70°, respectively, and arms folded across the chest.[6] The pelvis and torso were secured to the chair and the padded arm of the dynamometer was secured to the lower leg, approximately 3 cm proximal to the lateral malleolus and adjusted to align the knee joint axis of rotation with the dynamometer axis of rotation. The torque signal was output to an analog-to-digital converter (16-bit NI USB-6221; National Instruments, Austin, TX), sampled at 2000 Hz, and displayed in real time on a 56-cm computer screen using a custom-built software program (LabVIEW; National Instruments).

Participants performed a series of submaximal contractions, followed by 3 to 5 practice maximal-effort trials, during which participants were instructed to extend their knee as fast and with as much force as possible.[6] Participants performed practice trials, with at least 60 seconds of rest between each, until the maximum torque value from the trial was within 10% of the previous trial.[24] The average of the 3 greatest practice trials was used as a torque threshold that was displayed on a computer screen, with an additional target line set to 120% of the torque threshold.[24] The torque signal was provided in real time, and participants were instructed to extend their knee with enough effort to reach the target line (120% MVIC) during the 2 maximal-effort trials. During each maximal-effort trial, torque production was required to exceed the torque threshold line to ensure maximal effort was achieved.[24] Maximal-effort trials demonstrating a countermovement, in which the participant flexed the knee prior to extension, were discarded and repeated.

A second custom-built LabVIEW program was used to analyze quadriceps strength and RTD. Torque data were corrected for baseline passive torque production resulting from the weight of the limb prior to knee extension, and filtered using a fourth-order, zero-phase-shift, low-pass Butterworth filter with a cutoff frequency of 150 Hz.[6] Identification of torque onset was defined manually by an experienced investigator using a high-resolution x-axis and y-axis, as recommended by Maffiuletti et al.[25]

Briefly, a horizontal line was plotted 3 standard deviations above the baseline torque signal while the participant was at rest. This was used to guide the manual determination of where the torque signal deflected upward from baseline at the initial point of torque generation. A vertical cursor was placed at the point where the signal deflected from baseline (involved-limb preonset slope, –1.18 ± 4.4 Nm; contralateral-limb preonset slope, –1.53 ± 3.1 Nm).[6,14]

Quadriceps strength was defined as the data point corresponding to the peak torque achieved in the trial and normalized to body mass (Newton meters per kilogram).[24] Quadriceps RTD was defined as the slope of the torque-time curve, and was calculated over 3 time intervals, including (1) 0 to 50 milliseconds ($RTD_{0\text{-}50\ ms}$), (2) 100 to 200 milliseconds ($RTD_{100\text{-}200\ ms}$), and (3) peak RTD (RTD_{Peak}). This was determined as the peak derivative of the torque-time curve from onset to peak torque.[6,14] Quadriceps MVIC and RTD outcomes were averaged across the 2 maximal-effort testing trials and used for analysis.

Quadriceps MVIC and RTD were calculated for the involved and contralateral limbs. Bilateral quadriceps weakness has been previously reported in individuals with knee OA compared to age-matched control participants without knee OA.[8,13] Therefore, the researchers calculated average bilateral quadriceps MVIC and RTD outcomes from both limbs to determine the effect of bilateral quadriceps function on disability.

Assessment of Self-reported Disability and Physical Performance

Western Ontario and McMaster Universities Osteoarthritis Index Self-reported disability was assessed using the total score of the function subscale of the WOMAC, which is a reliable and valid assessment in individuals with tibiofemoral OA.[10] Scores from each item were summed and divided by the maximum possible score for function (maximum score, 68) to create a normalized WOMAC function score. Higher WOMAC scores indicate greater functional deficits.[10]

20-Meter Fast-Paced Walk Two sets of cones were placed 20 m apart in a hallway, and participants were instructed to walk as quickly as possible from one set of cones to the other and to continue walking through the finish line before stopping.[33] A stopwatch was used to record the time interval between when the participant initiated movement and when the participant passed the finish line.[33] The distance walked was divided by the average time to complete 3 trials to calculate walking speed (meters per second). A faster walking speed was interpreted as greater physical performance.

Chair-Stand Test A standard chair with a 47-cm seat height was used for the chair-stand test. Participants started the test seated, with arms crossed over the chest, and were instructed to rise to a full stand (ie, body erect and straight) and return to the initial seated position

as many times as possible in 30 seconds, while keeping their arms crossed.[17] The total number of completed chair stands was averaged across 3 trials and used for analysis. A greater number of completed chair-stand repetitions was interpreted as better physical performance.

Stair-Climb Test The stair climb was administered on a single flight of 10 steps, with a rise of 19 cm per step. Participants began at the bottom of the stairs and were instructed to ascend the stairs, turn around at the top of the flight, and descend as quickly as possible.[19] A stopwatch was used to record the time from when the participant initiated foot movement until both feet returned to the floor. The average time to complete the 3 trials was recorded in seconds and used for analysis. A shorter time to complete the stair climb was interpreted as better physical performance.

Statistical Analysis

The researchers evaluated disability and quadriceps RTD data for outliers, defined as greater than 3 standard deviations from the mean, using stem-and-leaf plots. If a statistical outlier was determined for a single outcome measure, then that participant was removed from the analysis for that variable only. This was done to preserve statistical power. First, the researchers evaluated age, sex, BMI, and the presence of bilateral radiographic OA (defined as a K-L grade of 2 or greater in each limb) as potential demographic covariates. The authors used a univariate linear regression model to determine whether each of these potential covariates was significantly associated with any outcome of disability (WOMAC, 20-m walk, chair stand, stair climb) or quadriceps RTD.

Next, as quadriceps strength is associated with self-reported function and physical performance in individuals with tibiofemoral OA,[26,32] the researchers used separate univariate linear regression models to determine the unique association between each measure of quadriceps RTD and each measure of disability after accounting for any significant demographic covariates and quadriceps strength. Therefore, significant demographic covariates were first entered into each model, followed by quadriceps strength, and then quadriceps RTD. The change in R^2 and the beta coefficient for RTD were determined after accounting for demographic covariates and quadriceps strength.

The authors completed separate analyses for quadriceps RTD intervals in the involved limb and the bilateral average between limbs. Associations were interpreted from the beta coefficient as weak (0.49 or less), moderate (0.5-0.69), and strong (0.7 or greater).[27] Significance was set a priori at $P \leq .05$ for all analyses, and analyses were performed using SPSS Version 21 (IBM Corporation, Armonk, NY).

RESULTS

A total of 420 individuals were screened by phone to determine study eligibility, based on the inclusion and exclusion criteria described above. Of those screened by phone, 121 individuals were scheduled for a laboratory testing session. Of those who participated in the laboratory testing session, 26 were excluded based on deficits in voluntary activation greater than 90%, 10 were excluded based on BMI greater than 35 kg/m^2, 2 were excluded based on K-L grades less than 2, 3 were excluded for WOMAC function score less than 31, and 4 were excluded based on previous medical history. Therefore, 76 participants with radiographic tibiofemoral OA completed testing (**TABLE 1**).

No statistical outliers were identified for any measure of disability. Three statistical outliers were identified for involved-limb $RTD_{0\text{-}50\ ms}$, and 2 were identified for bilateral average $RTD_{0\text{-}50\ ms}$. Quadriceps RTD was only assessed in the involved limb of 1 participant; therefore, the bilateral average between limbs was calculated for 75 participants.

Body mass index was found to be significantly associated with the 20-meter walk test ($R^2 = 0.13$, $\beta = -0.36$, $P = .001$), and therefore was added to the regression model as a covariate for this variable. No other demographic variables were found to be significantly associated with any other measure of disability or quadriceps RTD.

Involved Limb

Greater involved-limb $RTD_{100\text{-}200\ ms}$ was uniquely associated with faster walking speed (change in $R^2 = 0.05$, $\beta = 0.31$, $P = .013$). No other involved-limb RTD outcomes were uniquely associated with any outcome of disability after accounting for the involved-limb quadriceps strength (**TABLE 2**). Greater quadriceps strength was uniquely associated with lower WOMAC function scores (change in $R^2 = 0.15$, $\beta = -0.39$, $P<.001$), faster walking speed (change in $R^2 = 0.28$, $\beta = 0.57$, $P<.001$), greater number of chair stands (change in $R^2 = 0.21$, $\beta = 0.46$, $P<.001$), and faster time to complete the stair climb (change in $R^2 = 0.31$, $\beta = -0.56$, $P<.001$) (**TABLE 2**).

Bilateral Average

Greater bilateral average $RTD_{0\text{-}50\ ms}$ was uniquely associated with faster walking speed (change in $R^2 = 0.05$, $\beta = 0.23$, $P = .043$). Greater bilateral average $RTD_{100\text{-}200\ ms}$ was uniquely associated with faster walking speed (change in $R^2 = 0.20$, $\beta = 0.50$, $P<.001$) and faster time to complete the stair climb (change in $R^2 = 0.11$, $\beta = -0.41$, $P = .001$). Greater bilateral average RTD_{Peak} was uniquely associated with faster walking speed (change in $R^2 = 0.17$, $\beta = 0.46$, $P<.001$), greater number of chair-stand repetitions (change in $R^2 = 0.09$, $\beta = 0.33$, $P = .009$), and faster time to complete the stair climb (change in $R^2 = 0.11$, $\beta = -0.37$, $P = .003$). No other bilateral average RTD outcomes were uniquely associated with any outcome of disability after accounting for bilateral average quadriceps strength (**TABLE 3**).

Greater bilateral average quadriceps strength was uniquely associated with lower WOMAC function score (change in $R^2 = 0.17$, $\beta = -0.41$, $P<.001$), faster

walking speed (change in R^2 = 0.10, β = 0.30, P = .004), greater number of chair-stand repetitions (change in R^2 = 0.07, β = 0.27, P = .021), and faster time to complete the stair climb (change in R^2 = 0.08, β = −0.28, P = .016) (**TABLE 3**).

DISCUSSION

Most of this study's measures of quadriceps RTD in the involved limb exhibited a weak association with measures of physical performance in individuals with radiographic and symptomatic tibiofemoral OA. Specifically, greater bilateral average quadriceps RTD was moderately associated with faster walking speed. The bilateral average RTD during the late interval following torque onset (ie, $RTD_{100\text{-}200\ ms}$) exhibited a stronger association with faster walking speed and greater stair-climb performance compared to RTD during the early interval (ie, $RTD_{0\text{-}50\ ms}$) and overall RTD (RTD_{Peak}). No measure of quadriceps RTD was uniquely associated with the WOMAC function score after accounting for quadriceps strength. These data suggest that greater quadriceps RTD was associated with better physical performance, but not self-reported disability, in individuals with tibiofemoral OA, even after accounting for quadriceps strength.

Like the results of other studies assessing quadriceps RTD in individuals with total knee arthroplasty,[41] greater quadriceps RTD was found to be uniquely associated with greater walking speed, faster stair-climb performance, and greater number of chair stands completed in individuals with radiographic tibiofemoral OA (change in R^2 range, 0.05-0.20). Winters et al[41] demonstrated that 1 month following total knee arthroplasty, quadriceps RTD significantly predicted 12% and 15% of the variance in timed up-and-go and stair-climb performance, respectively, after accounting for quadriceps strength. At 6 months following total knee arthroplasty, quadriceps RTD continued to significantly predict 10% of the variance in physical performance, even though strength had improved to preoperative levels.[41] While different from RTD, muscle power (ie, product of force and velocity)[30] during concentric muscle contraction has been found to significantly predict a small percentage (change in R^2 range, 0.06-0.21) of performance outcomes in individuals with knee OA.[2,31,37] The present study's results, combined with results from previous studies,[2,31,37,41] suggest that the ability to generate torque quickly contributes a significant but small amount of additional variance to better physical performance in individuals with tibiofemoral OA, in addition to quadriceps strength.

The bilateral average during late-interval RTD (ie, bilateral average $RTD_{100\text{-}200\ ms}$) exhibited a moderate unique association with walking speed (change in R^2 = 0.20, β = 0.50), whereas the bilateral average during early RTD (bilateral average $RTD_{0\text{-}50\ ms}$ change in R^2 = 0.05, β = 0.23)

TABLE 1 Participant Demographics*

Characteristic	Value
Sex, n (%)	
Female	42 (55.3)
Male	34 (44.7)
Age, y	61.8 ± 7.1
BMI, kg/m^2	28.3 ± 4.0
Involved-limb K-L grade, n	
2	23
3	42
4	11
Contralateral-limb K-L grade, n	
0	6
1	15
2	25
3	20
4	3
Not applicable	7
Measures of disability	
Normalized WOMAC function score	30.9 ± 14.1
20-meter walk, m/s	1.8 ± 0.4
Chair stand, n	11.3 ± 4.9
Stair climb, s	16.3 ± 12.3
Involved-limb quadriceps function	
Quadriceps strength, Nm/kg	1.3 ± 0.5
$RTD_{0\text{-}50\ ms}$, Nm/s	85.0 ± 135.5
$RTD_{100\text{-}200\ ms}$, Nm/s	273.3 ± 210.8
RTD_{Peak}, Nm/s	1050.1 ± 716.9
Bilateral average quadriceps function	
Quadriceps strength, Nm/kg	1.4 ± 0.5
$RTD_{0\text{-}50\ ms}$, Nm/s	56.0 ± 85.4
$RTD_{100\text{-}200\ ms}$, Nm/s	237.7 ± 151.7
RTD_{Peak}, Nm/s	1164.2 ± 710.0

Abbreviations: BMI, body mass index; K-L, Kellgren-Lawrence; RTD, rate of torque development; WOMAC, Western Ontario and McMaster Universities Osteoarthritis Index.
**Values are mean ± SD unless otherwise indicated.*

TABLE 2 Involved-Limb Regression Analysis Results

Predictor Variable	Regression Model	Total *r*	R^2	R^2 Change	R^2 Change *P* Value	Beta Coefficient
$RTD_{0\text{-}50\ ms}$ (n = 73)						
WOMAC function						
Involved quadriceps strength	1	0.40	0.16	0.16	<.001	−0.40
Involved quadriceps strength, $RTD_{0\text{-}50\ ms}$	2	0.41	0.17	0.01	.688	−0.05
20-m walk						
BMI	1	0.37	0.14	0.14	.001	−0.37
BMI, involved quadriceps strength	2	0.64	0.40	0.26	<.001	0.54
BMI, involved quadriceps strength, $RTD_{0\text{-}50\ ms}$	3	0.65	0.42	0.02	.203	0.13
Chair stand						
Involved quadriceps strength	1	0.45	0.20	0.20	<.001	0.45
Involved quadriceps strength, $RTD_{0\text{-}50\ ms}$	2	0.46	0.21	0.01	.714	0.04
Stair climb						
Involved quadriceps strength	1	0.55	0.30	0.30	<.001	−0.57
Involved quadriceps strength, $RTD_{0\text{-}50\ ms}$	2	0.53	0.31	0.01	.353	−0.10
$RTD_{100\text{-}200\ ms}$ (n = 76)						
WOMAC function						
Involved quadriceps strength	1	0.39	0.15	0.15	<.001	−0.39
Involved quadriceps strength, $RTD_{100\text{-}200\ ms}$	2	0.41	0.17	0.02	.247	−0.17
20-m walk						
BMI	1	0.36	0.13	0.13	.001	−0.36
BMI, involved quadriceps strength	2	0.64	0.41	0.28	<.001	0.57
BMI, involved quadriceps strength, $RTD_{100\text{-}200\ ms}$	3	0.68	0.46	0.05	.013*	0.31
Chair stand						
Involved quadriceps strength	1	0.46	0.21	0.21	<.001	0.46
Involved quadriceps strength, $RTD_{100\text{-}200\ ms}$	2	0.46	0.21	0.00	.973	0.01
Stair climb						
Involved quadriceps strength	1	0.56	0.31	0.31	<.001	−0.56
Involved quadriceps strength, $RTD_{100\text{-}200\ ms}$	2	0.57	0.32	0.01	.202	−0.17
RTD_{Peak} (n = 76)						
WOMAC function						
Involved quadriceps strength	1	0.39	0.15	0.15	<.001	−0.39
Involved quadriceps strength, RTD_{Peak}	2	0.41	0.17	0.02	.231	−0.17
20-m walk						
BMI	1	0.36	0.13	0.13	.001	−0.36
BMI, involved quadriceps strength	2	0.64	0.41	0.28	<.001	0.57
BMI, involved quadriceps strength, RTD_{Peak}	3	0.66	0.44	0.03	.06	0.22
Chair stand						
Involved quadriceps strength	1	0.46	0.21	0.21	<.001	0.46
Involved quadriceps strength, RTD_{Peak}	2	0.46	0.21	0.00	.759	0.04
Stair climb						
Involved quadriceps strength	1	0.56	0.31	0.31	<.001	−0.56
Involved quadriceps strength, RTD_{Peak}	2	0.57	0.32	0.01	.296	−0.13

Abbreviations: BMI, body mass index; RTD, rate of torque development; WOMAC, Western Ontario and McMaster Universities Osteoarthritis Index.
**Significant association between RTD outcome and measure of disability.*

TABLE 3 Bilateral Average Regression Analysis Results

Predictor Variable	Regression Model	Total *r*	R^2	R^2 Change	R^2 Change *P* Value	Beta Coefficient
$RTD_{0\text{-}50\ ms}$ (n = 73)						
WOMAC function						
Bilateral quadriceps strength	1	0.40	0.16	0.16	<.001	−0.40
Bilateral quadriceps strength, $RTD_{0\text{-}50\ ms}$	2	0.40	0.16	0.00	.551	0.07
20-m walk						
BMI	1	0.36	0.13	0.13	.002	−0.36
BMI, bilateral quadriceps strength	2	0.48	0.23	0.10	.003	0.32
BMI, bilateral quadriceps strength, $RTD_{0\text{-}50\ ms}$	3	0.52	0.27	0.05	.043*	0.23
Chair stand						
Bilateral quadriceps strength	1	0.27	0.07	0.07	.022	0.27
Bilateral quadriceps strength, $RTD_{0\text{-}50\ ms}$	2	0.33	0.11	0.04	.091	0.21
Stair climb						
Bilateral quadriceps strength	1	0.28	0.08	0.08	.017	−0.28
Bilateral quadriceps strength, $RTD_{0\text{-}50\ ms}$	2	0.34	0.12	0.04	.08	−0.21
$RTD_{100\text{-}200\ ms}$ (n = 75)						
WOMAC function						
Bilateral quadriceps strength	1	0.41	0.17	0.17	<.001	−0.41
Bilateral quadriceps strength, $RTD_{100\text{-}200\ ms}$	2	0.41	0.17	0.00	.681	−0.06
20-m walk						
BMI	1	0.36	0.13	0.13	.001	−0.36
BMI, bilateral quadriceps strength	2	0.47	0.23	0.10	.004	0.30
BMI, bilateral quadriceps strength, $RTD_{100\text{-}200\ ms}$	3	0.65	0.42	0.20	<.001*	0.50
Chair stand						
Bilateral quadriceps strength	1	0.27	0.07	0.07	.021	0.27
Bilateral quadriceps strength, $RTD_{100\text{-}200\ ms}$	2	0.33	0.10	0.04	.056	0.24
Stair climb						
Bilateral quadriceps strength	1	0.23	0.08	0.08	.016	−0.28
Bilateral quadriceps strength, $RTD_{100\text{-}200\ ms}$	2	0.44	0.19	0.11	.001*	−0.41
RTD_{Peak} (n = 75)						
WOMAC function						
Bilateral quadriceps strength	1	0.41	0.17	0.17	<.001	−0.41
Bilateral quadriceps strength, RTD_{Peak}	2	0.43	0.18	0.01	.321	−0.15
20-m walk						
BMI	1	0.36	0.13	0.13	.001	−0.36
BMI, bilateral quadriceps strength	2	0.47	0.23	0.10	.004	0.30
BMI, bilateral quadriceps strength, RTD_{Peak}	3	0.64	0.40	0.17	<.001*	0.46
Chair stand						
Bilateral quadriceps strength	1	0.27	0.07	0.07	.021	0.27
Bilateral quadriceps strength, RTD_{Peak}	2	0.39	0.16	0.09	.009*	0.33
Stair climb						
Bilateral quadriceps strength	1	0.23	0.08	0.08	.016	−0.28
Bilateral quadriceps strength, RTD_{Peak}	2	0.43	0.19	0.11	.003*	−0.37

Abbreviations: BMI, body mass index; RTD, rate of torque development; WOMAC, Western Ontario and McMaster Universities Osteoarthritis Index.
**Significant association between RTD outcome and measure of disability.*

exhibited a weak association with walking speed. Differences in the physiological contributions to early- and late-interval RTD may partially explain these findings. Late-interval RTD is associated with structural and compositional factors of the muscle such as muscle quality (ie, ultrasound-measured echogenicity), pennation angle, and cross-sectional area.[14] Therefore, individuals with compromised muscle composition and smaller quadriceps cross-sectional area may demonstrate lower late-interval RTD.[14]

Lower late-interval RTD may contribute to slower walking speed and stair-climb performance. Conversely, early-interval RTD reflects neural activation rather than structural contributors to muscle function.[30,38] Individuals with knee OA demonstrate alterations in motor-unit activation patterns of the quadriceps, including activation of larger motor units and the number of active motor units recruited.[21] Altered neuromuscular activation may limit the ability to generate RTD early following torque onset and place a greater reliance on maximal muscle strength to complete functional tasks of daily living, such as fast-paced walking and stair climbing.

Involved-limb (change in $R^2 = 0.05$, $\beta = 0.31$) quadriceps $RTD_{100\text{-}200\ ms}$ exhibited a weak association with walking speed. Interestingly, the bilateral average quadriceps $RTD_{100\text{-}200\ ms}$ demonstrated a significant, moderate association with walking speed (change in $R^2 = 0.20$, $\beta = 0.50$). Declines in muscle function occur in both the involved and contralateral limbs in individuals with tibiofemoral OA.[8,13,34] As most functional tasks require bilateral contribution from the quadriceps, bilateral deficits in quadriceps RTD may have influenced physical performance to a greater extent than the involved limb alone.

Additionally, the presence of bilateral tibiofemoral OA was not significantly associated with self-reported function or physical performance in this study's cohort. Therefore, bilateral deficits in quadriceps strength and RTD may contribute to diminished physical performance compared to unilateral deficits in the involved limb, regardless of a bilateral OA diagnosis. These findings suggest that rehabilitation implemented bilaterally in patients with tibiofemoral OA, regardless of the presence of unilateral or bilateral radiographic OA, may be more beneficial for improving physical performance compared to only treating the symptomatic limb.

Most of this study's quadriceps RTD outcomes demonstrated significant, yet weak, associations with the selected measures of physical performance (**TABLES 2** and **3**). The combination of quadriceps strength and RTD did, however, result in a significant moderate association with walking speed and stair-climb performance (**TABLE 3**). These results support previous literature demonstrating the association between greater quadriceps strength and less disability in individuals with knee OA.[26,32] In the current study, greater involved-limb quadriceps strength was significantly associated with self-reported disability at all 3 physical performance assessments (β range, −0.36 to −0.56).

Moreover, the combination of greater quadriceps strength and greater RTD may result in better physical performance. The combination of quadriceps strength and $RTD_{100\text{-}200\ ms}$ resulted in the strongest association with walking speed (total $r = 0.65$) (**TABLE 3**) compared to quadriceps strength or RTD alone, suggesting that both strength and RTD contribute to physical performance in individuals with tibiofemoral OA. While improving both quadriceps strength and RTD may be beneficial for improving physical performance in individuals with tibiofemoral OA, improving muscle strength should still be a predominant focus of rehabilitation.

Specifically targeting quadriceps strength as well as quadriceps RTD through therapeutic exercise may be beneficial for limiting disability. Previous work has concluded that strength training (ie, high resistance, slow movement speed) and power training (ie, low resistance, fast movement speed) produce similar improvements in neuromuscular function in older adults.[40] Others have suggested that older adults demonstrate greater improvements in strength and RTD in addition to performance in activities, such as the chair-stand test, following power training.[22] Explosive-contraction strength training has been demonstrated to improve both early and late RTD time intervals, whereas conventional strength training only results in improvements during late RTD time intervals.[3] Further research is needed to identify the most effective intervention strategies for improving both quadriceps strength and quadriceps RTD in both the affected and contralateral limbs, as well as how changes in quadriceps RTD may influence disability in individuals with tibiofemoral OA.

While quadriceps RTD was associated with physical performance (ie, walking speed, stair climb), quadriceps RTD was not associated with self-reported disability as quantified by the WOMAC function subscale. Whereas this study's physical performance measures require a large demand from the quadriceps for successful completion, the WOMAC assesses self-reported pain and functional limitations across a wide range of activities of daily living that place various demands on the quadriceps. A high level of quadriceps RTD is likely not utilized in all tasks assessed by the WOMAC, such as "putting on/taking off socks," which may have resulted in the negligible association between quadriceps RTD and WOMAC function score. Further research is needed to determine whether additional neuromuscular factors are associated with self-reported disability and physical performance in individuals with tibiofemoral OA. However, quadriceps strength was significantly associated with self-reported disability (change in $R^2 = 0.15$, $\beta = -0.39$) (**TABLE 2**). As such, improving quadriceps strength may have an overall greater impact on reducing self-reported disability compared to only focusing on RTD.

Limitations

While this study provides insight into neuromuscular mechanisms that may contribute to disability in individuals with tibiofemoral OA, it is important to acknowledge its limitations to better inform future research. The authors assessed quadriceps strength and RTD during a non–weight-bearing isometric contraction that isolated the quadriceps. Generalized lower extremity muscle weakness, rather than quadriceps weakness specifically, might have influenced the ability to perform functional tasks, as all performance assessments (fast-paced walk, chair stand, stair climb) required the function of multiple muscles and actions from multiple joints.

Pain also may have influenced the ability to perform functional tasks; however, the authors did not assess the level of pain during the strength or performance assessments in this study.

Further, the researchers utilized a cross-sectional design, prohibiting them from determining the causal nature of the association between quadriceps RTD and physical performance in individuals with tibiofemoral OA. Future studies should evaluate whether improvements in quadriceps RTD are associated with improvements in physical performance and disability.

The authors assessed the contribution of various measures of quadriceps RTD to self-reported function and physical performance. Multiple comparisons may increase the chance of committing a type I error.

The researchers also limited their enrollment to individuals with radiographic and symptomatic tibiofemoral OA demonstrating deficits in neuromuscular activation (central activation ratio less than 90%). It is possible that the low associations between early RTD and physical performance outcomes in this study are only generalizable to a cohort of individuals with a central activation ratio less than 90%. It remains unclear how these results would translate to the larger population of individuals with tibiofemoral OA who demonstrate various degrees of functional deficits and muscle activation deficits.

Additionally, muscle strength and power both decline with age,[1,14] and it is unknown how much of the changes in quadriceps strength and RTD was due to normal aging or the OA disease process.

CONCLUSION

Measures of quadriceps RTD were found to be weakly associated with disability after accounting for quadriceps strength in individuals with tibiofemoral OA. Bilateral average quadriceps RTD, assessed during the late interval following torque onset, exhibited a stronger association with physical performance compared to the involved limb independently. However, no measure of quadriceps RTD was significantly associated with self-reported disability. Further research is needed to determine whether an improvement in quadriceps RTD results in better physical performance in individuals with tibiofemoral OA. ◉

KEY POINTS

FINDINGS: Involved-limb quadriceps rate of torque development (RTD) was weakly associated with physical performance outcomes, but not self-reported disability, in persons with tibiofemoral osteoarthritis. Bilateral average quadriceps RTD was moderately associated with faster walking speed.

IMPLICATIONS: Greater bilateral quadriceps RTD may have a small influence on physical performance in individuals with tibiofemoral osteoarthritis.

CAUTION: These results are limited by the cross-sectional nature of this study and the assessment of RTD during a non–weight-bearing contraction. It remains unknown how a change in quadriceps RTD is related to a change in physical performance, and how RTD assessed during other contraction types may also influence physical performance.

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Association of Hip and Foot Factors With Patellar Tendinopathy (Jumper's Knee) in Athletes

Patellar tendinopathy is an overuse injury associated with jumping and landing activities[2] and thought to be caused by excessive or repetitive forces applied to the patellar tendon.[15] The prevalence of this condition in elite volleyball and basketball athletes has been reported to be about 40%,[23] and prevention and management strategies are often challenging.[9] Understanding the risk profile of this condition is necessary because patellar tendinopathy is associated with long-term pain and functional limitations that can not only affect sport participation but also end a sport career.[9]

BACKGROUND: Investigations on the causes of patellar tendinopathy should consider impairments at the hip and foot/ankle because they are known to influence movement patterns and affect patellar tendon loading.

OBJECTIVES: To investigate hip and foot/ankle impairments associated with patellar tendinopathy in volleyball and basketball athletes using classification and regression tree analysis.

METHODS: In this clinical measurement, cross-sectional study, 192 athletes were assessed for impairments of the hip and foot/ankle, including shank-forefoot alignment, dorsiflexion range of motion (ROM), iliotibial band flexibility, passive hip internal rotation ROM, and hip external rotator and hip abductor isometric strength. Athletes with tenderness and/or pain at the inferior pole of the patella were considered to have patellar tendinopathy. Athletes with scores higher than 95 points on the Victorian Institute of Sport Assessment-patella (VISA-P), no pain during the single-leg decline squat, and no history of patellar tendon pain were considered not to have patellar tendinopathy. Classification and regression tree analyses were performed to identify interacting factors associated with patellar tendinopathy.

RESULTS: Interactions among passive hip internal rotation ROM, shank-forefoot alignment, and hip external rotator and abductor strength identified athletes with and without patellar tendinopathy. The model achieved 71.2% sensitivity and 74.4% specificity. The area under the receiver operating characteristic curve was 0.77 (95% confidence interval: 0.70, 0.84; $P<.001$).

CONCLUSION: Impairments of the hip and foot/ankle are associated with the presence of patellar tendinopathy in volleyball and basketball athletes. Future studies should evaluate the role of these impairments in the etiology of patellar tendinopathy.
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KEY WORDS: *decision trees, epidemiology, knee, sports, tendon injury*

The majority of studies evaluating factors related to patellar tendinopathy have focused on anthropometric,[8,14,43] biomechanical, and anatomical variables[35,37,41] that are frequently related to the knee joint (local factors).[14,17,28] A systematic review published by van der Worp et al[41] found weak associations between local, nonlocal, and demographic risk factors and patellar tendinopathy. Thus, it is possible that other factors, such as those related to impairments of the hip and foot/ankle, could play a role in patellar tendon overload and, consequently, contribute to patellar tendinopathy.

It has been suggested that impairments of the hip and foot/ankle may contribute to the development of pathological conditions of the knee by means of direct and indirect influences on movement patterns or anatomical alignment that could overload the knee structures.[1,7,12,13,18,26,29-31,36,37,39,41] For example, there is evidence that individuals with patellofemoral pain have increased hip adduction[12] and excessive femoral internal rotation (IR)[39] during functional activities. These altered hip motions may be caused by hip abductor and hip external rotator (ER) weakness.[44] In addition, studies investigating

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lower-limb kinematics in the sagittal plane reported that athletes with patellar tendinopathy exhibit altered hip and knee movement patterns during landing, when compared to those who are pain free.[25,35,38]

However, no investigation has included analysis of factors related to frontal and transverse planes of motion in individuals with patellar tendinopathy. It has been reported that altered hip joint range of motion (ROM) during landing tasks could overload the patellar tendon.[25] Moreover, deficits in ankle dorsiflexion (DF) ROM and increased varus foot alignment also have been found to lead to excessive foot pronation and increased lower-limb IR,[40] which are thought to overload the patellar tendon.[1,26,34] Thus, the presence of hip abductor and ER weakness, ROM impairments (restriction or excessive mobility), and anatomical alignment at the hip joint and foot-ankle complex may influence lower extremity movement patterns and, consequently, alter the force distribution on the knee joint structures (eg, patellar tendon).[5,11,12,19,26,37,41]

Because of the complexity related to injury occurrence, statistical methods that include linear and nonlinear associations and the identification of interaction effects should be used.[4,16,32] The classification and regression tree (CART) method allows the identification of factors and uncovers interactions related to a specific condition.[20] This method has been used in the medical literature to identify clinical decision rules that enable the classification of participants into clinically important categories.[20] In addition, this statistical method may reveal the complex relationships among factors related to impairments of the hip and foot/ankle that may contribute to patellar tendinopathy. Thus, the objective of this cross-sectional study was to investigate, by means of CART analysis, impairments of the hip and foot/ankle that are associated with patellar tendinopathy in volleyball and basketball athletes.

METHODS

Participants

Male and female volleyball and male basketball athletes from professional clubs in Brazil participated in a preseason screening over an 8-month period. The inclusion criteria were regular sport participation of at least 12 hours per week during the immediate previous season, absence of Osgood-Schlatter disease and absence of anterior knee pain not related to the patellar tendon (both confirmed by a sport medicine physician or physical therapist), and no history of lower-limb surgery and/or patellar tendon steroid injection (as determined by self-report of the athlete or by the physical therapist). All participants read and signed the informed-consent form approved by the Universidade Federal de Minas Gerais Ethics in Research Committee (report number 0493.0.203.000-09).

Athletes with patellar tendinopathy were those with tenderness and/or pain at the inferior pole of the patella (confirmed by a sport medicine physician or physical therapist). The severity of symptoms was assessed using the Victorian Institute of Sport Assessment-patella (VISA-P) questionnaire. This tool is scored from 0 to 100, with a score of less than 80 points indicating severe patellar tendinopathy (n = 59).[11,42] A VISA-P score greater than 95 points, no pain during the single-leg decline squat, and no patellar tendon pain history indicated that an athlete did not have patellar tendinopathy (n = 133).[22,27,45] Those athletes with VISA-P scores between 80 and 94 points were excluded from the study.[42]

Clinical Assessment

Preseason assessments included shank-forefoot alignment (SFA), DF ROM, iliotibial band flexibility, passive hip IR ROM, and hip ER and hip abductor isometric strength. These tests were selected because the conditions they evaluate are thought to contribute to excessive femoral and tibial motions in frontal and transverse planes.[1,5,7,12,19,24,26,29,39,44] The clinical measurements were made sequentially, with the strength assessments performed last. Six pilot studies were conducted to determine each measurement's reliability by means of intraclass correlation coefficient (ICC). Two examiners with at least 5 years of experience in preseason assessments were trained in all tests to obtain acceptable ICC values (**TABLE 1**).

Both examiners participated in more than 300 assessments prior to the current study, which contributed to their excellent reliability coefficients and, consequently, small standard error of the measurement (SEM) and minimal detectable difference values for all variables. Different samples were used in each of the pilot studies.

TABLE 1 Pilot Study Sample Size, Intrarater and Interrater Reliability, SEM, and MDD of Each Variable Examined

Variable	Sample Size, n	Interval Between Measures, s	Intrarater Reliability*	Interrater Reliability*	SEM	MDD
SFA, deg	10	4	0.93	0.90	2.47	6.82
Hip ER torque, Nm	6	7	0.98	0.90	0.53	1.49
Hip abductor torque, Nm	6	7	0.94	0.90	1.10	3.05
Ankle DF ROM, deg	12	4	0.98	0.92	0.56	1.57
Passive hip IR ROM, deg	6	4	0.99	0.99	0.55	1.52
Iliotibial band flexibility, deg	6	4	0.99	0.94	0.31	0.85

Abbreviations: DF, dorsiflexion; ER, external rotator; IR, internal rotation; MDD, minimal detectable difference; ROM, range of motion; SEM, standard error of the measurement; SFA, shank-forefoot alignment.
**Values are intraclass correlation coefficient.*

Shank-forefoot alignment and ankle DF ROM required a larger sample due to low variability of the measurements previously identified in other studies. **TABLE 1** shows the sample size, interval between measures, intrarater and interrater reliability, SEM, and minimal detectable difference of each variable.

The SFA was measured according to the method described by De Michelis Mendonça et al,[10] with the participant lying prone on a treatment table. The shank was bisected by a line joining the midpoint of the tibial plateau and the midpoint between the medial and lateral malleoli. A metal rod was strapped over the metatarsophalangeal heads. The participant actively maintained neutral position of the ankle joint (90°), and the examiner took a picture of foot position with a digital camera (D5000; Nikon Corporation, Tokyo, Japan). The SFA was determined, using the Simi Motion Twinner software (Simi Reality Motion Systems GmbH, Unterschleissheim, Germany), as the angle between the shank bisection line and the orientation of the metal rod positioned on the forefoot (**FIGURE 1A**). Positive values indicated varus alignment.[10] The mean of the SFA, determined from 3 photos, was used for analysis.

A handheld dynamometer (microFET2; Hoggan Scientific, LLC, Salt Lake City, UT) was used to assess hip ER and abductor isometric strength.[26,44] Hip ER strength was measured with the participant positioned in prone, with the pelvis stabilized with a rigid strap. The handheld dynamometer was placed proximal to the medial malleolus,[44] and the participant performed 3 isometric contractions (**FIGURE 1B**). A 15-second interval was provided between trials. Force values were multiplied by the linear distance from the dynamometer placement to the hip axis of rotation and normalized by body mass (Newton meters per kilogram).

Hip abductor strength was assessed using the protocol described by Bittencourt et al,[5] with the participant positioned in sidelying and the pelvis stabilized with a rigid strap. The dynamometer was positioned proximal to the lateral femoral condyle. Participants performed 3 trials of maximal isometric hip abduction contractions with a 15-second interval between trials (**FIGURE 1C**). Hip abduction torque was calculated as the product between the mean of 3 force measures and the distance from the greater trochanter to the location of the dynamometer.

Ankle DF ROM was assessed using the protocol described by Bennell et al.[3] The participant was positioned facing a wall and was instructed to move the knee forward until it touched the wall. The participant maintained the foot on this line without lifting the heel off the floor (**FIGURE 2A**). An analogical inclinometer (Starrett, Athol, MA) was placed 15 cm from the tibial tuberosity to define maximum shank anterior inclination (ankle DF ROM). The average of 3 measurements was considered for analysis.[1]

To evaluate passive hip IR ROM, the examiner applied the protocol described by Carvalhais et al,[6] in which the participant was positioned prone on a treatment table with the knee flexed to 90°. The passive movement of hip IR, produced by the weight of the leg and foot, was allowed until tension in muscle and passive structures of the hip joint stopped this movement (**FIGURE 2B**). In other words, the end position was one in which the torque produced by the mass of the lower leg and foot was equal to the passive-resistance torque generated to prevent further hip IR. In contrast to typical passive ROM measurements, this test did not rely on the examiner's perception of the hip joint end feel. Prior to the test, the examiner moved the hip joint passively to produce the tissue's viscoelastic accommodation.[6] The passive hip IR ROM was measured with an analog inclinometer (Starrett) positioned 5 cm from the tibial tuberosity, and the mean of 3 measures (degrees) was used for analysis.[6] This particular method of passive hip IR ROM was selected because it is related to hip passive stiffness.[6] A previous study reported excellent ICC (0.99) and small SEM (1.5°) values for this measure.[5]

The modified Ober test protocol was used to assess iliotibial band flexibility, as described by Reese and Bandy.[33] The participant was positioned in sidelying, with the arms in front of the body. The examiner maintained the pelvis alignment with one hand (keeping the pelvis in neutral rotation relative to the treatment table) and performed passive hip joint motions of flexion, external rotation, abduction, and extension. The examiner then removed the support slowly until the lower limb moved to the position of maximal hip adduction. A second examiner measured the thigh's inclination to the horizontal (hip adduction angle)

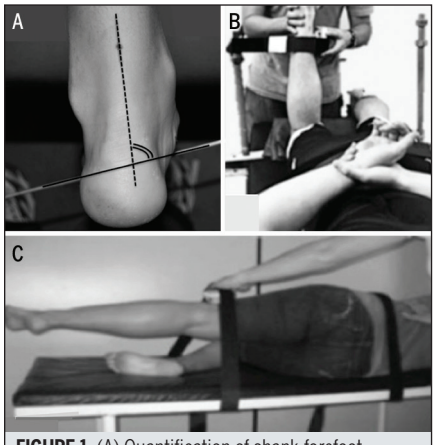

FIGURE 1. (A) Quantification of shank-forefoot alignment angle with 2-dimensional software, (B) position for hip external rotation, and (C) hip abductor isometric strength assessment.

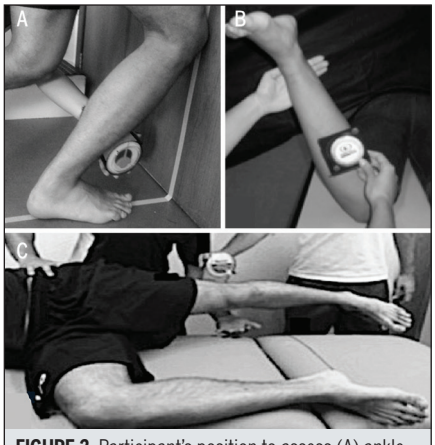

FIGURE 2. Participant's position to assess (A) ankle dorsiflexion range of motion, (B) passive hip internal rotation range of motion, and (C) iliotibial band flexibility.

with an analog inclinometer (Starrett) positioned distal to the lateral femoral condyle (**FIGURE 2C**). Before the test, the examiner simulated the test 3 times to allow the tissue's viscoelastic accommodation. Measures were taken 3 times, and the mean (degrees) was used for analysis. Positive values were considered as hip abduction (less flexible iliotibial band).[26]

Participants were assessed bilaterally, and all examiners who performed the preseason screening tests were blinded to the participant's group assignment. Data from the injured or most symptomatic lower limb for the athletes with patellar tendinopathy, and from the dominant lower limb in athletes without patellar tendinopathy, were considered for analysis.[5] Limb dominance was identified by asking the participants which leg they would choose to kick a ball.

Statistical Analysis

Descriptive statistics of VISA-P questionnaire score, age, height, weight, sex, iliotibial band flexibility, hip abductor torque, hip ER torque, passive hip IR ROM, ankle DF ROM, and SFA were used to characterize the sample. A CART analysis was used to determine which factors and interactions were associated with the presence or absence of patellar tendinopathy.[20] The CART is a multivariate and nonparametric classification model that, throughout binary recursive divisions of the initial set of data, selects the predictors and their respective cutoff points that best classify the individuals in each of the outcome categories,[20] in this case, presence or absence of patellar tendinopathy. The predictors are selected based on the strength of association with the outcome variable (patellar tendinopathy occurrence), and the divisions reveal interactions among predictors. The CART model starts with all data (n = 192) in the first node. Then, the variable that is most associated with the outcome is selected, and the data are divided into 2 groups, according to specific cutoff values. For all nodes, the CART model searches for the best cutoff value for partitioning the selected variable to get the maximum separation of the sample in terms of patellar tendinopathy occurrence (presence or absence of patellar tendinopathy). This process produces a classification similar to a tree.

The following criteria were used to produce the partitions and, consequently, tree growth: a minimum of 8 participants in each node to make a division, a minimum of 4 participants to generate a node, and a Gini index of 0.0001 to maximize the node's homogeneity. A maximum depth of 3 levels was established, and a pruning procedure was applied to avoid overfitting partitions.[5] The classification cost was considered symmetric between categories, and patellar tendinopathy occurrence probability was established as equal between groups.[23]

After CART model development, a receiver operating characteristic (ROC) curve was created to verify the accuracy of the model.[5] A probability of type I error of .05 was used to verify whether the area under the ROC curve was different from 0.5, which indicates that the model was accurate to predict the outcome categories. Finally, prevalence ratios were calculated for each terminal node of the CART model to investigate the strength of associations.

RESULTS

Descriptive Data

Initially, 311 athletes participated in the preseason screening. Of those, 41 were excluded due to the presence

TABLE 2 Descriptive Demographic and Independent Variable Data of the Entire Sample, Separated by Those With and Without Patellar Tendinopathy *

Variable	Range	Total Sample (n = 192)	With Patellar Tendinopathy (n = 59)	Without Patellar Tendinopathy (n = 133)	Excluded (n = 80)
Age, y	15-37	17.85 ± 4.72	18.25 ± 0.69	17.68 ± 0.38	16.71 ± 2.99
Time in practice, y	1-20	5.12 ± 4.64	5.71 ± 0.62	4.86 ± 0.39	5.20 ± 3.57
Height, m	1.53-2.13	1.86 ± 0.10	1.85 ± 0.01	1.86 ± 0.09	1.84 ± 0.11
Body mass, kg	51.40-125	76.47 ± 13.58	75.32 ± 1.88	76.56 ± 1.26	75.25 ± 14.66
BMI, kg/m^2	16.18-30.37	21.73 ± 2.48	21.70 ± 0.29	21.79 ± 0.24	22.02 ± 2.83
VISA-P score	39-100	90.32 ± 14.83	68.73 ± 1.14	99.70 ± 0.08	90.75 ± 7.78
Ankle DF ROM, deg	23.66-54.33	39.52 ± 6.26	39.48 ± 0.79	39.89 ± 0.58	38.87 ± 6.43
Passive hip IR ROM, deg	10.97-59.71	32.92 ± 10.6	34.49 ± 10.04	32.25 ± 10.90	34.04 ± 11.03
Iliotibial band flexibility, deg	−16.2-14.98	3.91 ± 4.80	4.27 ± 4.48	3.74 ± 4.94	4.89 ± 3.91
Hip ER torque, Nm/kg	0.09-0.81	0.33 ± 0.13	0.30 ± 0.01	0.35 ± 0.01	0.35 ± 0.15
Hip abductor torque, Nm/kg	0.58-2.65	1.51 ± 0.36	1.49 ± 0.05	1.50 ± 0.03	1.49 ± 0.35
SFA, deg	−3.68-46.64	22.31 ± 9.22	23.38 ± 1.16	21.93 ± 0.87	18.46 ± 10.90

Abbreviations: BMI, body mass index; DF, dorsiflexion; ER, external rotator; IR, internal rotation; ROM, range of motion; SFA, shank-forefoot alignment; VISA-P, Victorian Institute of Sport Assessment-patella.
**Values are mean ± SD unless otherwise indicated.*

of Osgood-Schlatter disease, history of surgery, or steroid injection. Using the criteria described above, 59 athletes were included in the patellar tendinopathy group (22 women and 37 men) and 133 in the non–patellar tendinopathy group (25 women and 108 men). Participants with a VISA-P score between 80 and 94 points (n = 78) were excluded from this study. **TABLE 2** shows descriptive statistics for demographic characteristics and predictors for the study sample. Statistical difference of descriptive variables between athletes with and without patellar tendinopathy was found only for the VISA-P questionnaire score ($P<.001$), which was expected given the inclusion criteria.

CART Model and Prevalence Ratios

The classification tree identified passive hip IR ROM, SFA, hip ER torque, and hip abductor torque as predictors for patellar tendinopathy (**FIGURE 3**). Passive hip IR ROM was the first predictor selected by the CART model, with a cutoff of 40.76°. In individuals with lower values of passive hip IR ROM, SFA was the second predictor, with a cutoff point of 16.95°, and hip ER torque was selected as the third predictor, with a cutoff point of 0.31 Nm/kg. For individuals with passive hip IR ROM values above the cutoff point, the model selected another passive hip IR ROM cutoff point (44.46°) on the second level, and hip abductor torque with a cutoff point of 1.57 Nm/kg on the third level.

The model indicated that the interaction of lower values of passive hip IR ROM with lower values of SFA was best at predicting the absence of patellar tendinopathy (terminal node 3). The absence of patellar tendinopathy also was observed in individuals with lower values of passive hip IR ROM and greater values of forefoot varus alignment, but with greater values of hip ER torque (terminal node 8). On the other hand, patellar tendinopathy occurrence was best predicted by passive hip IR ROM between 40.76° and 44.46° (terminal node 5) and by the interaction of lower passive hip IR ROM, greater SFA, and lower hip ER torque (terminal node 7). The interactions representing the patellar tendinopathy presence/absence profiles are illustrated in **FIGURE 4**.

TABLE 3 provides the prevalence ratios for each terminal node and the strength of the associations of predictors with outcome. The results indicated that the interactions among predictors of nodes 3, 5, 7, and 8 were statistically associated with the absence or presence of patellar tendinopathy.

The CART model correctly predicted 42 of the 59 athletes with patellar tendinopathy (71.2% sensitivity) and 99 of the 133 athletes without patellar tendinopathy (74.4% specificity). The total prediction of the model was 73.4%, and the area under the ROC curve was 0.77

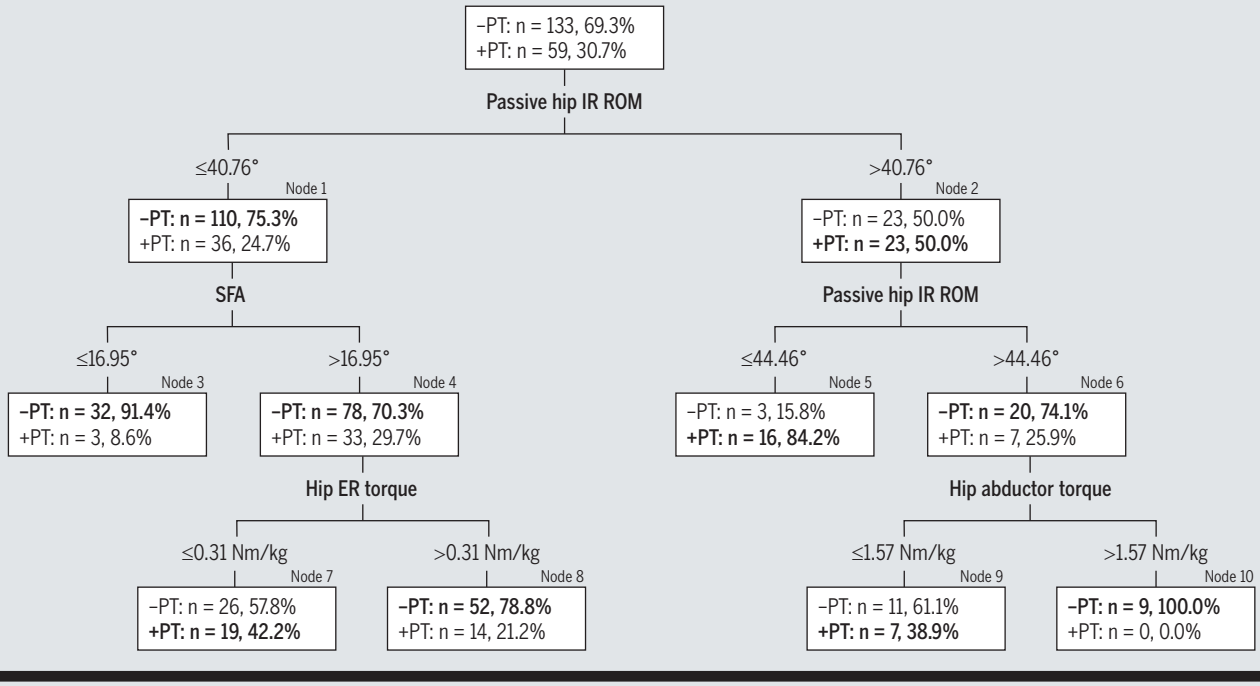

FIGURE 3. Classification and regression tree model for PT. The bold text in each node (+PT or –PT) corresponds to the predicted category. The classification profile for the presence of PT in terminal node 5 was passive hip IR ROM between 40.76° and 44.46°; in terminal node 7 was passive hip IR ROM under 40.76°, SFA above 16.95°, and hip ER torque under 0.31 Nm/kg; and in terminal node 9 was passive hip IR ROM above 44.46° and hip abductor torque under 1.57 Nm/kg. The classification profile for the absence of PT in terminal node 3 was passive hip IR ROM under 40.76° and SFA under 16.95°; in terminal node 8 was passive hip IR ROM under 40.76°, SFA above 16.95°, and hip ER torque above 0.31 Nm/kg; and in terminal node 10 was passive hip IR ROM above 44.46° and hip abductor torque above 1.57 Nm/kg. Abbreviations: ER, external rotation; IR, internal rotation; PT, patellar tendinopathy; ROM, range of motion; SFA, shank-forefoot alignment.

(95% confidence interval: 0.70, 0.84; standard error, 0.03; $P<.001$), indicating that the model's classification was not due to chance.

DISCUSSION

By means of CART analysis, the authors examined the interactions among clinical measurements associated with patellar tendinopathy.[4] The CART model revealed that interactions among variables related to the hip joint and foot-ankle complex were associated with the presence or absence of patellar tendinopathy. Specifically, different interactions among passive hip IR ROM, SFA, hip ER torque, and hip abductor torque were associated with patellar tendinopathy occurrence. Overall, the model accurately identified 71.2% of athletes with patellar tendinopathy and 74.4% of athletes without patellar tendinopathy. Thus, interactions among other variables not investigated in this study, including biomechanical, behavioral, and physiological factors, also may contribute to the occurrence of patellar tendinopathy and should be explored in future studies.

The first predictor selected by the CART model was passive hip IR ROM, with a cutoff point of 40.76°. Interestingly, hip IR ROM was associated with patellar tendinopathy occurrence or absence according to its cutoff points and the presence of other predictors (SFA and hip abductor and ER torques). Specifically, athletes with lower values of passive hip IR ROM (less than or equal to 40.76°, intermediary node 1), while having lower values of foot varus alignment (SFA less than or equal to 16.95°, terminal node 3), had 0.24 times the likelihood of having patellar tendinopathy (prevalence ratio of 0.24). In other words, they had a 76% less likelihood of having patellar tendinopathy. This finding is consistent with studies that have shown that athletes with adequate passive hip IR ROM (not too high or too low) and proper foot alignment have a decreased probability of exhibiting excessive lower-limb IR (and thus less patellar tendon loading) during weight-bearing tasks.[5,40]

Interestingly, athletes with lower passive hip IR ROM (less than or equal to 40.76°, intermediary node 1) but larger varus values of SFA (greater than 16.95°, intermediary node 4) had a 41% less likelihood of having patellar tendinopathy when they had greater values of hip ER torque (prevalence ratio of 0.59, terminal node 8). Although greater varus foot alignment may contribute to excessive pronation and excessive tibia IR,[40] the presence of adequate hip ER strength may help in controlling lower-limb IR, thereby mitigating the possible deleterious effects of this motion on the patellar tendon.[21,44]

The contribution of hip torque to the absence of patellar tendinopathy also was observed in terminal node 10. All athletes with greater values of passive hip IR ROM (greater than 44.46°, intermediary node 6), which is associated with low hip stiffness,[6] but greater values of hip abductor torque (greater than 1.57 Nm/kg, terminal node 10) were classified as not hav-

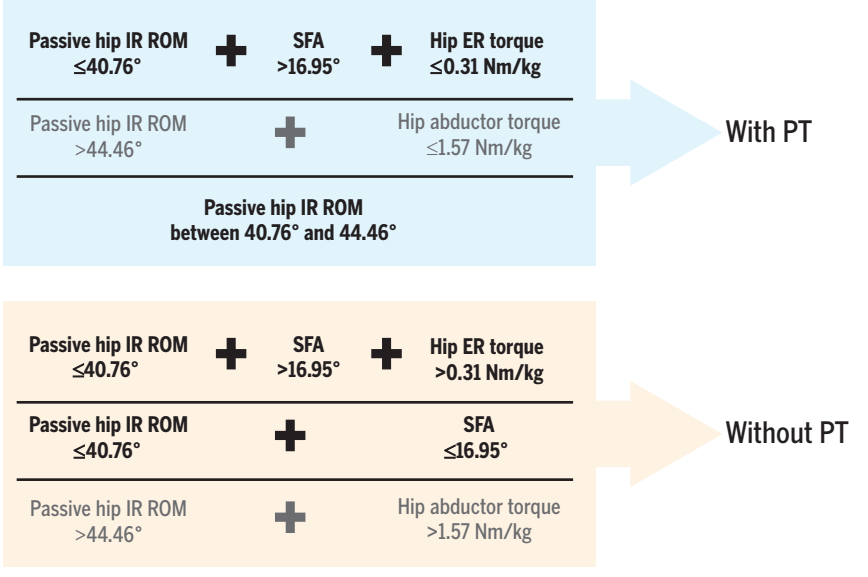

FIGURE 4. Interactions (profiles) revealed by the classification and regression tree model. Interactions in bold were statistically significant (see **TABLE 3**). Abbreviations: ER, external rotation; IR, internal rotation; PT, patellar tendinopathy; ROM, range of motion; SFA, shank-forefoot alignment.

TABLE 3 Prevalence Ratio of Each Terminal Node of the CART Model

Terminal Node	Prevalence Ratio*
3	0.24 (0.07, 0.72)†
5	3.38 (2.45, 4.68)†
7	1.55 (1.01, 2.39)†
8	0.59 (0.35, 0.99)†
9	1.30 (0.69, 2.42)
10‡	...

Abbreviation: CART, classification and regression tree.
**Values in parentheses are 95% confidence interval.*
†Statistically significant.
‡In terminal node 10, it was not possible to calculate the prevalence ratio because all athletes (100%) were classified as not having patellar tendinopathy.

ing patellar tendinopathy (n = 9; 100% did not have patellar tendinopathy). The authors speculate that the presence of adequate hip abductor strength could decrease patellar tendinopathy occurrence in athletes with high passive hip IR ROM.

Demonstrating the nonlinear and complex nature of factors associated with patellar tendinopathy, interactions among passive hip IR ROM, SFA, and hip torques also were associated with patellar tendinopathy presence, depending on their cutoff values. Athletes with lower passive hip IR ROM (less than or equal to 40.76°, intermediary node 1), larger varus SFA values (greater than 16.95°, intermediary node 4), and lower hip ER torque (less than or equal to 0.31 Nm/kg, terminal node 7) had a 55% increase in likelihood of having patellar tendinopathy (prevalence ratio of 1.55, terminal node 7). Lower levels of hip abductor torque (less than or equal to 1.57 Nm/kg, terminal node 9), in the presence of greater passive hip IR ROM (greater than 44.46°, intermediary node 6), also were related to patellar tendinopathy occurrence (prevalence ratio of 1.30, terminal node 9). This classification, however, did not achieve statistical significance with respect to prevalence ratio, and the interpretation that the interaction between low passive hip stiffness and hip abductor weakness may contribute to patellar tendinopathy occurrence should be made with caution.

The CART results indicated that individuals with passive hip IR ROM between 40.76° and 44.46° had a 238% increase in likelihood of developing patellar tendinopathy (prevalence ratio of 3.38, terminal node 5). As values of hip IR ROM over 40° may indicate low hip stiffness,[6] this result suggests that hip IR ROM values in this range may be enough to contribute to the occurrence of patellar tendinopathy, in the absence of other relevant risk or protective factors investigated in this study. However, it is important to note that, as a classification method, CART analysis may sometimes produce overly complex trees that do not generalize well from the analyzed sample (overfitting) and produce results that are difficult to interpret.[20] In addition, as the individuals in terminal node 5 had an important increase in the likelihood of having patellar tendinopathy, other factors not investigated in the present study may have contributed to the observed result. Although this result should be taken with caution, the contribution of low hip IR stiffness to patellar tendinopathy occurrence cannot be ignored.

These results should be interpreted with caution, as causal relationships cannot be established by the methods used (cross-sectional study). It is important to note that the prediction of this model be validated by an independent sample to verify the specificity and sensitivity of the model. That said, methodological issues should be considered when interpreting these results: (1) the cutoff points indicated are sample dependent, meaning that they may vary between populations and samples; and (2) prospective investigations need to be conducted to determine whether the physical impairments identified could be considered as a risk profile for patellar tendinopathy occurrence. In addition, it is necessary to investigate the contribution of movement impairments, apart from physical impairments, to patellar tendinopathy occurrence. A limitation of this study is that some of the participants were undergoing physical therapy at the time of testing, which might have affected the results. Moreover, factors such as trunk, quadriceps, and hamstrings strength and flexibility,[36,37,41] years of sport practice, and hours played,[2,17,43] although not the focus of the present study, should be investigated in the future.

The results of this study revealed nonlinear and complex interactions between predictors and the outcome variable and identified profiles related to patellar tendinopathy occurrence or absence. The contribution of one variable to the outcome (eg, passive hip IR ROM) depended on the presence of other variables (eg, SFA and hip torques). These results may help guide clinical practice, as clinicians might select interventions based on the individual profile established by the final CART nodes. For example, athletes with passive hip IR ROM under 40.76°, SFA above 16.95°, and hip ER torque under 0.31 Nm/kg have a greater chance of having patellar tendinopathy and, therefore, should undergo interventions focused on modifying these factors (eg, use of foot orthotics and hip ER strengthening). In addition, preventive programs could be planned based on achieving proper hip IR ROM (above 40.76°), adequate SFA alignment (under 16.95°), and high hip ER torque (above 0.31 Nm/kg), as athletes with this profile had increased likelihood of not having patellar tendinopathy.

CONCLUSION

Factors related to the hip joint and foot-ankle complex were associated with the presence or absence of patellar tendinopathy in volleyball and basketball athletes. The CART analysis produced a model that revealed interactions between passive hip IR ROM, SFA, hip ER torque, and hip abductor torque that should be considered when managing patellar tendinopathy–associated factors in athletes. ◉

KEY POINTS

FINDINGS: Impairments of the hip and foot/ankle were associated with the presence of patellar tendinopathy in volleyball and basketball athletes. Classification and regression tree analysis revealed that these factors interact to create a profile associated with patellar tendinopathy occurrence.

IMPLICATIONS: Clinicians may apply interventions or preventive programs based on the risk profile derived by the findings of this study. The interactions among factors are complex, and additional research is needed before the full implications of these results for clinical practice can be established.

CAUTION: The data of the present study were only collected during preseason evaluation in active athletes, some of

whom were undergoing physical therapy, which might have affected these results. The results of this study are preliminary, and the classification and regression tree (CART) model should be independently validated in a larger sample.

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Compensatory Strategies That Reduce Knee Extensor Demand During a Bilateral Squat Change From 3 to 5 Months Following Anterior Cruciate Ligament Reconstruction

BACKGROUND: Decreased extensor moments in the surgical knee during bilateral squats can persist beyond 1 year following anterior cruciate ligament reconstruction (ACLR). This is accomplished using interlimb and intralimb compensations.

OBJECTIVES: This study sought to assess loading during squatting longitudinally, 3 and 5 months post ACLR, and to determine the extent to which interlimb and intralimb compensations contribute to reduced knee extensor moments.

METHODS: In this controlled, longitudinal laboratory study, 11 individuals (4 male) underwent 3-D motion analysis of a squat at 3 and 5 months post ACLR. A repeated-measures multivariate analysis of variance (limb by time) assessed differences in peak knee and hip flexion angles, knee extensor moment, vertical ground reaction force, and hip-to-knee extensor moment ratio. Stepwise linear regression analysis was used to determine the contribution of interlimb (between-limb vertical ground reaction force ratio) and intralimb (within-surgical-limb hip-to-knee moment ratio) compensations to the between-limb knee extensor moment ratio.

RESULTS: A significant effect of limb was observed for knee flexion angle, knee extensor moment, vertical ground reaction force, and hip-to-knee extensor moment ratio, while a significant effect of time was observed for knee extensor moment and hip-to-knee extensor moment ratio. At 3 months, the vertical ground reaction force ratio and hip-to-knee extensor moment ratio predicted the knee extensor moment ratio (R^2 = 0.854, P<.001). At 5 months, the hip-to-knee extensor moment ratio predicted the knee extensor moment ratio (R^2 = 0.584, P = .006).

CONCLUSION: Individuals used interlimb and intralimb compensations to reduce the knee extensor moment of the surgical limb at 3 months post ACLR. Similar reductions in the knee extensor moment at 5 months were accomplished with only intralimb compensations. *J Orthop Sports Phys Ther 2018;48(9):713-718. Epub 12 Jun 2018. https://doi.org/10.2519/jospt.2018.7977*

KEY WORDS: *anterior cruciate ligament reconstruction, interlimb compensation, intralimb compensation, knee extensor moment deficit, rehabilitation*

Following anterior cruciate ligament reconstruction (ACLR), individuals may adopt loading patterns that shift the mechanical demands away from the surgical knee during bilateral tasks.[2,5,6,12,13] Given the presence of joint-level impairments such as pain, decreased strength, and range of motion following surgery, the adoption of loading strategies that underload the knee is not surprising. However, despite the emphasis on restoring knee loading during rehabilitation,[4,8] these strategies appear to persist over time.[3,9,10] At 13 months post ACLR, individuals exhibited average knee extensor moments that were 17% smaller in the surgical limb compared to the nonsurgical limb during a bilateral squat against body-weight resistance.[12] Asymmetry of this magnitude would not be expected during what would be considered a submaximal task at a time when individuals are returning or have returned to sports and high-level activities.[5,6,12] The persistence of underloading is concerning, as asymmetrical limb loading during landing tasks has been linked to increased risk for anterior cruciate ligament (ACL) reinjury.[11]

Bilateral squatting tasks are incorporated into early rehabilitation to progressively load the knee as individuals progress to more demanding single-limb

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tasks.[14] However, the bilateral multijoint nature of a squat allows for compensations that can shift the task demands to the nonsurgical limb (interlimb compensation) or to adjacent joints within the surgical limb (intralimb compensation) to reduce knee extensor moments. While no study has evaluated squat mechanics during early rehabilitation, Labanca et al[5] found that individuals 1 month post ACLR performed bilateral sit-to-stand tasks with a 38% reduction in vertical ground reaction forces (vGRFs) in the surgical limb. This suggests that during early recovery, individuals depend on the nonsurgical limb to reduce demands on the surgical knee during bilateral tasks.

Data evaluating squat mechanics at 1 to 2 years post ACLR suggest that individuals continue to reduce sagittal plane knee loading, not only by compensating with the contralateral limb, but also by shifting the demands within the limb to the hip.[12] Specifically, reduced knee extensor moments have been found along with increased hip extensor moments, in combination with[7] and in the absence of[13] between-limb differences in ground reaction forces, during squat tasks. Together, these studies suggest that individuals may rely on interlimb compensations to unload the knee during early rehabilitation but adopt intralimb compensations as they progress through rehabilitation. However, evaluation of squat performance over time is needed to determine whether a shift in compensatory strategies occurs.

Understanding the mechanisms used to underload the knee in the sagittal plane, particularly during rehabilitation, when bilateral squats are introduced and progressed, will inform clinicians and help develop strategies to prevent long-term persistence. The purpose of this study was to (1) assess loading patterns during a bilateral squat task in individuals longitudinally at 3 and 5 months post ACLR, and (2) determine how interlimb and intralimb compensations contribute to reduced knee extensor moments at these time points.

METHODS

Participants

Participants were included in the study if they were between the ages of 14 and 40 years, had undergone ACLR, and were currently receiving physical therapy treatment. They were excluded if they had a current injury to the contralateral limb that would influence function or had concurrent knee pathology that limited their weight-bearing status. Participants were recruited from a single physical therapy clinic, and the researchers did not control for specific interventions. However, 4 treating physical therapists agreed that their treatments followed standard rehabilitation protocols that emphasized early restoration of range of motion and progression off crutches when patients demonstrated gait without observable deviations.

Balance and lower extremity strengthening exercises were progressed based on the individual patient's ability.[1,15] Participants were assessed longitudinally at 2 time points: 3 and 5 months after surgery. All procedures were explained to each participant, and informed consent was obtained as approved by the Institutional Review Board of the University of Southern California Health Sciences Campus. Parental consent and youth assent were obtained for all participants under the age of 18 years.

Procedures

Testing took place at the Human Performance Laboratory in the Division of Biokinesiology and Physical Therapy at the University of Southern California, located at the Competitive Athletes Training Zone (Pasadena, CA). Kinematic data were collected using an 11-camera motion-capture system at 250 Hz (Qualisys AB, Gothenburg, Sweden). Ground reaction forces were obtained using two 1.20 × 0.60-m force platforms at 1500 Hz (Advanced Mechanical Technology, Inc, Watertown, MA). Kinematic and kinetic data were collected synchronously using Qualisys Track Manager Version 2.8 (Qualisys AB).

Prior to biomechanical testing, participants rode a stationary bike to warm up for 5 minutes before placement of the reflective markers. The reflective markers (25-mm spheres) were placed on the following anatomical landmarks to define body segments: the L5-S1 junction, and bilaterally on the end of second toes, first and fifth metatarsal heads, medial and lateral malleoli, medial and lateral epicondyles of femurs, greater trochanters, posterior superior iliac spines, and iliac crests. Reflective markers were affixed to the participant's skin using 5-mm circular double-sided tape and reinforced with Transpore tape (3M, St Paul, MN). Tracking marker clusters mounted on semi-rigid plastic plates were secured to the thighs, shanks, and heel plates of shoes using flexible fabric bands and duct tape. A static calibration trial was collected with all markers attached. Tracking marker clusters, as well as markers on the end of the second toe, iliac crests, posterior superior iliac spines, and the L5-S1 junction, remained on the participant throughout the entire testing, while other markers were removed after the static calibration trial.

Participants were asked to perform squats with each foot on an individual force platform, with feet shoulder-width apart and arms crossed over the chest. No additional weight or resistance was added. Participants were instructed to squat down as low as possible without pain and to return to the standing position between each squat. After practice, they performed 2 trials of 5 consecutive squats at a self-selected comfortable pace. A 3-minute break between each trial was provided to prevent fatigue. The self-selected rate of squatting (squats per second) was calculated to determine whether it differed between individuals.

Data Analysis

Marker coordinate data were labeled and digitized using Qualisys Track Manager Version 2.8 (Qualisys AB). Lower extrem-

ity kinematics and net joint moments were calculated using Visual3D (C-Motion, Inc, Germantown, MD) software. Data were filtered using a fourth-order, zero-lag, 12-Hz Butterworth low-pass filter. Local coordinate systems of the pelvis, thighs, shanks, and feet were derived from the standing calibration trial, and the 6 degrees of freedom of each segment were determined from the segment's triad of reflective markers. Ankle, knee, and hip sagittal plane kinematics were calculated by determining the transformation from the triad of markers to the position and orientation of each segment determined from the standing calibration trial, using a joint coordinate system approach. Body mass was calculated by dividing the average vGRF over 5 seconds during the static trial by 9.81 N/kg. Body mass, kinematics, and ground reaction force were used to calculate sagittal plane net joint moments at the knee and hip, using standard inverse dynamics equations. Moments, reported as internal moments, were normalized to body mass.

Peak sagittal plane moments at the knee and hip, peak vGRF, and peak knee and hip flexion angles were identified during the deceleration phase of the squat. The deceleration phase was defined as the time from standing (highest position of L5-S1 marker) to peak knee flexion angle. To characterize knee extensor moment deficits in the surgical knee, a ratio of peak knee extensor moments was calculated between limbs (ie, surgical limb divided by nonsurgical limb). To characterize the distribution of forces between the limbs, a between-limb ratio of peak vGRFs was calculated in the same way. For these ratios, 1 indicates symmetry between limbs, less than 1 indicates that the nonsurgical limb is greater than the surgical limb, and greater than 1 indicates that the surgical limb is greater than the nonsurgical limb.

To characterize the relative contributions of hip and knee moments to deceleration, a within-limb ratio of hip extensor moment to knee extensor moment was calculated for each limb. For this ratio, 1 indicates symmetry between hip and knee extensor moments within the limb, less than 1 indicates that the knee moment is greater than the hip moment, and greater than 1 indicates that the hip moment is greater than the knee moment. Variables were averaged across 3 repetitions (middle 3 out of 5 repetitions) from 2 trials for each participant.

Statistical Analysis

Descriptive statistics (mean, standard deviation, and Cohen's *d* effect size) were calculated for all variables. The rate of squatting was compared across time using a paired *t* test. To determine whether differences exist between limbs or across time in peak knee and hip extensor moments, peak vGRF, peak knee and hip flexion angles, and within-limb hip-knee ratio during deceleration, a 2-way (limb by time) repeated-measures multivariate analysis of variance was performed. In the case of a main effect of limb or time, paired *t* tests were performed.

Separate stepwise linear regressions were used to determine the independent contribution of interlimb (vGRF ratio) and intralimb (hip-knee moment ratio) compensations to knee extensor moment deficits (between-limb knee extensor moment ratio) in the surgical limb at each time point (3 and 5 months). The criterion of probability of F to enter of .05 or less was used to enter a variable into the model, and the criterion of probability of F to remove of .10 or greater was used to remove a variable from the model. Significance levels were set at $\alpha \leq .05$. All analyses were performed using PASW Statistics Version 18 (IBM Corporation, Armonk, NY).

RESULTS

Eleven eligible individuals (mean ± SD age, 22.9 ± 9.5 years; 7 female, 4 male; height, 171.5 ± 9.7 cm; weight, 67.5 ± 10.1 kg) participated in this longitudinal observational study. Allograft (n = 4) and bone-patellar tendon-bone (n = 6) and hamstring (n = 1) autograft procedures were performed by 4 different surgeons. Seven participants reported concurrent injuries: meniscal tear (n = 6) and medial collateral ligament injury (n = 1). None of these concurrent injuries were treated surgically. None of the participants reported weight-bearing restrictions following surgery. Three participants reported previous ACLR to the contralateral (n = 2) and ipsilateral knees (n = 1) at least 2 years prior to reinjury, and all had returned to presurgical levels of physical activity.

Descriptive statistics (mean ± SD) are reported in the **TABLE**. No significant difference in the rate of squatting was observed over time ($P = .598$). No significant limb-by-time interaction was observed, while a main effect of limb was present ($F = 6.59$, $P = .028$). Post hoc paired *t* tests were performed to compare between limbs, collapsed across time for each variable of interest. Compared to the nonsurgical limb, the surgical limb exhibited a significantly lower peak knee flexion angle (2.4° ± 2.6°; $P = .013$; effect size, 0.95), lower peak knee extensor moment (0.37 ± 0.18 Nm/kg; $P<.001$; effect size, 2.16), less peak vGRF (0.09 ± 0.06 N/kg; $P<.001$; effect size, 1.69), and greater hip-knee ratio (0.68 ± 0.55; $P = .002$; effect size, 1.28) (**FIGURE**). No significant difference was noted between limbs for hip flexion angle ($P = .504$) and extensor moment ($P = .383$).

At 3 months, the between-limb vGRF ratio and within-limb hip-knee extensor moment ratio (surgical limb) together predicted the knee extensor moment ratio ($R^2 = 0.854$, $P<.001$). The vGRF ratio entered the regression equation first and was the largest predictor of the knee extensor moment ratio ($R^2 = 0.624$, $P = .004$, $\beta = 0.790$). The hip-knee extensor moment ratio entered second ($R^2 = 0.230$, $P = .007$, $\beta = -0.492$). Smaller vGRF ratios and larger hip-knee extensor moment ratios predicted smaller knee extensor moment ratios. At 5 months, the hip-knee extensor moment ratio (surgical limb) was the only variable to enter the regression equation ($R^2 = 0.584$, $P =$

.006, $\beta = -0.765$). Larger hip-knee extensor moment ratios predicted smaller knee extensor moment ratios.

DISCUSSION

Substantial deficits in knee extensor moments were observed in the surgical limb at both time points. On average, a 38% decrease in knee extensor moment was observed in the surgical compared to the nonsurgical knee at 3 months. While the deficits appear to decrease to 30% at 5 months post ACLR, the absence of a significant interaction between limb and time suggests that these deficits did not significantly improve over time. Studies have reported deficits of smaller magnitudes at later time points following surgery, suggesting that further improvements are likely past 5 months, but their persistence at any magnitude during submaximal tasks is of concern.[5,12] Moreover, the underloading strategies reported in this study are present at a time when rehabilitation is focused on more demanding single-limb exercises and progression to running.

Differences between limbs with respect to vGRF and the hip-knee extensor moment ratio at 3 and 5 months suggest that both between- and within-limb compensations are present. On average, vGRF was 13% lower under the surgical compared to the nonsurgical limb, indicating that individuals were underloading their surgical limb. The hip-knee extensor moment ratio was, on average, 40% larger in the surgical limb, indicating that individuals shifted the extensor demands away from the knee and toward the hip in the surgical limb. At 3 and 5 months post ACLR, the hip-knee extensor moment ratio in the nonsurgical limb was close to 1, indicating that peak hip and knee extensor moments were relatively equal during the squat. In contrast, the hip-knee extensor moment ratio in the surgical limb was well above 1 at both time points, indicating that the peak hip extensor moment was larger than the knee extensor moment. The absence of a limb-by-time interaction suggests that these deficits were not different between 3 and 5 months post ACLR.

However, further analyses of their contributions to knee extensor moment deficits at each time point suggest that the underlying mechanics for underloading the knee may differ with time. At 3 months post surgery, both vGRF ratio and surgical-limb hip-knee extensor moment ratio predicted knee extensor moment deficits. Together, they accounted for 85% of the variance in the between-limb knee extensor moment ratio. The vGRF ratio, however, was the largest predictor, explaining 62% of the variance. The hip-knee extensor moment ratio accounted for the remaining 23%. Lower vGRF and larger hip-knee extensor moment ratios predicted smaller knee extensor moments in the surgical limb. These data suggest that at 3 months post ACLR, individuals appear to utilize both interlimb and intralimb compensations but rely more heavily on an interlimb compensation as they shift their weight to the opposite limb. At 5 months post ACLR, only the hip-knee extensor moment ratio predicted knee extensor moment deficits in the surgical limb, indicating that individuals preferentially utilized an intralimb compensation to underload the knee extensors at 5 months. Despite the similarity in the magnitude of knee extensor moment deficits in the surgical limb, it appears that individuals alter the underlying compensatory strategy from 3 to 5 months following surgery.

Interestingly, this change in strategy was observed in the absence of significant

TABLE Descriptive Statistics*

Variable/Time	Surgical Limb	Nonsurgical Limb
Peak knee flexion, deg		
3 mo	101.5 ± 12.9†	103.7 ± 12.6
5 mo	104.3 ± 14.8	106.7 ± 15.0
Peak hip flexion, deg		
3 mo	92.1 ± 17.7	91.1 ± 18.6
5 mo	88.5 ± 15.9	88.6 ± 16.0
Peak knee extensor moment, Nm/kg		
3 mo	0.67 ± 0.24†	1.08 ± 0.18
5 mo	0.78 ± 0.22	1.11 ± 0.25
Peak hip extensor moment, Nm/kg		
3 mo	1.11 ± 0.24	1.05 ± 0.22
5 mo	1.11 ± 0.35	1.06 ± 0.36
Peak vertical ground reaction force, N/kg		
3 mo	0.58 ± 0.08†	0.68 ± 0.06
5 mo	0.62 ± 0.09	0.70 ± 0.12
Hip-knee extensor moment ratio		
3 mo	1.81 ± 0.68†	1.00 ± 0.26
5 mo	1.50 ± 0.60	0.96 ± 0.28
Peak vertical ground reaction force between-limb ratio		
3 mo	0.86 ± 0.11	
5 mo	0.89 ± 0.07	
Rate of squatting, squats/s		
3 mo	0.57 ± 0.04	
5 mo	0.55 ± 0.06	

*Values are mean ± SD.
†Significant main effect of limb.

changes in hip extensor moments or vGRFs over time. Only the contribution of these variables to knee extensor moment deficits changed over time. The absence of a significant contribution from the vGRF ratio at 5 months suggests that individuals may become more comfortable loading the surgical limb at 5 months, leaving only the intralimb compensation to explain underloading of the knee.

These findings have several implications for rehabilitation following ACLR. Substantial decreases in knee extensor moments were observed during the performance of a submaximal task at a time of rehabilitation when patients are expected to be capable of meeting the mechanical demands of the task. Loading impairments may be difficult to identify clinically, as they were observed along with small differences between limbs in peak knee flexion and no differences between limbs in peak hip flexion. This presents a clinical challenge, given that between-limb differences in knee angle as small as 3° are difficult to detect clinically. During early rehabilitation, strategies for restoring symmetrical weight bearing during bilateral tasks should be emphasized and reinforced even during submaximal tasks. In addition, efforts should be made to continue to focus on sagittal plane knee loading and avoid compensation with the hip extensors. These efforts may be more important in later rehabilitation. Finally, efforts must be made to identify mechanisms for identifying these deficits in the clinic.

While these data longitudinally describe knee sagittal plane underloading strategies following ACLR at 2 rehabilitation time points, interpretation of the data in the present study may be limited due to the small sample size recruited. Two participants included in the analyses had previous contralateral ACLR. Given that ratio calculations compare the surgical limb to what is considered a healthy nonsurgical limb, unresolved impairments may influence the ratio calculations, making these individuals appear less impaired. However, analyses of these data without the 2 participants revealed the same results, suggesting that they were not behaving differently.

Determination of factors that underlie underloading of the knee in the sagittal plane cannot be made based on the current data. In the absence of strength data, it is not known whether individuals can meet the mechanical demands of the task. The fact that the tasks were performed within a self-selected, pain-free range and that there were no complaints of pain suggests that pain did not factor heavily into task performance. No electromyography was collected, thus limiting the authors' ability to determine the presence of hamstring cocontraction during the task. If present, then knee extensor moments may have been underestimated, as inverse dynamics calculate net joint moments. Surgical reconstruction techniques, surgeons, and treating physical therapists varied across participants, making it difficult to attribute deficits or compensatory strategies to a type of surgery or rehabilitation. Despite the large and medium effect sizes observed, a larger sample size would allow for improved generalization to a typical population of patients treated in a physical therapy clinic.

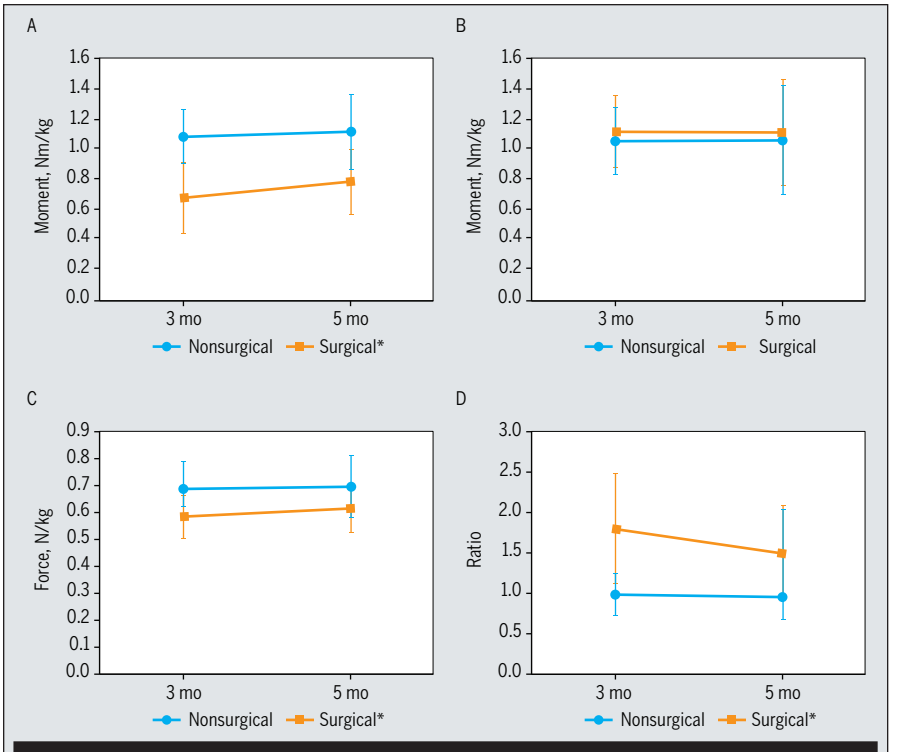

FIGURE. Mean ± SD (A) peak knee extensor moment, (B) peak hip extensor moment, (C) peak vertical ground reaction force, and (D) hip-knee extensor moment ratio between surgical (orange line) and nonsurgical (blue line) limbs during the deceleration phase of a bilateral squat at 3 and 5 months post anterior cruciate ligament reconstruction. *Main effect of limb.

CONCLUSION

Substantial deficits in knee extensor moments in the surgical limb exist at 3 and 5 months post ACLR during a squat against body-weight resistance to a self-selected depth. These deficits do not improve between 3 and 5 months, but the compensatory strategies that contribute to reduced knee extensor moments change across this time interval. At 3 months, individuals relied more heavily on a between-limb compensation, shifting weight to the other limb while also shifting the demand to the hip

extensors within the surgical limb. By 5 months post ACLR, decreased knee extensor moments in the surgical limb were primarily driven by an intralimb shift to the hip extensors. Despite a difference between limbs in vGRF, a shift away from the nonsurgical limb did not explain knee extensor moment deficits at 5 months post ACLR.

KEY POINTS

FINDINGS: After anterior cruciate ligament reconstruction, individuals exhibited deficits in sagittal plane loading of the knee during bilateral squats at 3 and 5 months following surgery. While these deficits did not improve during this time, the strategy used to compensate for reduced knee extensor moments shifted from a combined interlimb and intralimb compensatory strategy at 3 months to an intralimb strategy at 5 months.

IMPLICATIONS: During rehabilitation of individuals following anterior cruciate ligament reconstruction, efforts should be made to encourage equal distribution of loads between the limbs and avoid compensation with the hip extensors during bilateral squats. An increased focus on the restoration of knee sagittal plane loading during double-limb tasks in early rehabilitation may be needed to limit the persistence of underloading after rehabilitation.

CAUTION: This study did not determine which factors underlie impairments in knee extensor moments. The effectiveness of any recommendations for improving such impairments is speculative.

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Hand-Grip Strength: Normative Reference Values and Equations for Individuals 18 to 85 Years of Age Residing in the United States

BACKGROUND: Hand-grip strength is an indicator of overall strength and a predictor of important outcomes. Up-to-date, population-specific reference values for measurements of grip strength are needed to properly interpret strength outcomes.

OBJECTIVES: To provide population-based grip-strength reference values and equations for US residents 18 to 85 years of age.

METHODS: Hand-grip data from 1232 participants 18 to 85 years of age were extracted from the database of the 2011 normative phase of the US National Institutes of Health Toolbox project in this cross-sectional study. Descriptive reference values and equations were derived from the data.

RESULTS: The authors present grip-strength reference values using summary statistics (mean, standard deviation, and percentile). The mean grip strength ranged from 49.7 kg for the dominant hand of men 25 to 29 years of age to 18.7 kg for the nondominant hand of women 75 to 79 years of age. The researchers also present reference regression equations for the dominant and nondominant sides of men and women. The explanatory variables in the equations are age, height, and weight.

CONCLUSION: The normative reference values and equations provided in this study may serve as a guide for interpreting grip-strength measurements obtained from tested individuals. *J Orthop Sports Phys Ther 2018;48(9):685-693. Epub 23 May 2018. doi:10.2519/jospt.2018.7851*

KEY WORDS: *grip, hand, hand strength, NIH Toolbox (health care), norm*

Published research provides extensive support for the measurement of hand-grip strength. That research shows grip strength to be (1) an acceptable indicator of overall muscle strength[5]; (2) impaired in various musculoskeletal,[1,3] neuromuscular,[10] and cardiovascular/pulmonary conditions[3,4]; (3) fundamental to the diagnosis of frailty[18] and sarcopenia[12]; and (4) prognostic of untoward outcomes such as mortality and future disability.[4] Grip strength has been recommended for routine use as a vital sign[4] and as a screening tool in primary care[22] and hospital practice.[21]

The identification of grip-strength impairments requires normative reference values to which an individual's grip-strength measurements can be compared. There are numerous peer-reviewed studies that provide such values for populations outside the United States. These populations include those of Great Britain,[13] Germany,[20] Spain,[23] Australia,[24] Canada,[41] Korea,[35] Taiwan,[42] and Japan.[33] Studies[25,26,28] that provide grip-strength reference values have also been conducted in the United States. Unfortunately, most of these studies[25,26] were completed before 1986 and used small convenience samples. Ideally, normative reference values should have been obtained within the past 15 to 20 years[38] from a population-representative sample.[31]

There are 2 large-scale, relatively recent studies involving the population of the United States that provide grip-strength data over the lifespan and so might be used to generate normative reference values. One study, the National Health and Nutrition Examination Survey (NHANES),[11] is an ongoing research program conducted by the US Centers for Disease Control and Prevention and National Center for Health Statistics to assess the health and nutritional status of adults and children in the United States and to track changes over time. Perna et al[28] reported grip-strength estimates using

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2011-2012 NHANES data, but the authors looked at the strength of both hands combined. The other study is the US National Institutes of Health (NIH) Toolbox.[19] It incorporates a multidimensional set of brief, royalty-free measures for assessing emotional, cognitive, sensory, and motor function in people aged 3 to 85 years. Data were collected for the normative phase of the NIH Toolbox project in 2011,[2] but up to this point, normative data have only been published in the peer-reviewed literature for individuals 3 to 17 years of age.[8]

The authors of the present study compared grip-strength outcomes and procedures of the NHANES and NIH Toolbox studies.[9] Stratified grip-strength values derived from the data of the 2 studies were sometimes comparable, but the instrumentation and procedures used in these studies differed considerably; those of the NIH Toolbox were much more consistent with the recommendations of the American Society of Hand Therapists[17] and Roberts et al.[32] Those recommendations stipulate that the second handle position of the dynamometer be used when evaluating grip strength. Patients should be seated with the shoulder adducted and neutrally rotated, the elbow flexed at 90°, and the forearm and wrist in neutral position. Thus, data from the NIH Toolbox study were used to address the 2 purposes of the present study: (1) to provide US population-based grip-strength reference values for individuals 18 to 85 years of age, and (2) to develop normative regression equations for grip strength.

METHODS

NIH Toolbox Project

The NIH Toolbox project is a 4-year study designed to develop an assessment battery that provides a standard set of royalty-free, brief, and comprehensive assessment tools for use by researchers and clinicians in a variety of settings, with a particular emphasis on measuring outcomes in longitudinal, epidemiological, and prevention or intervention clinical research across the lifespan of individuals 3 to 85 years of age. Grip strength was identified in 2007 as a core construct for inclusion in the NIH Toolbox motor domain, based on literature and instrument database reviews, requests for information, consensus meetings, and expert interviews.

The NIH Toolbox team completed a validation study in 2009 of 340 healthy participants.[7,30,39,40] Using the standardized testing protocol, the validation study assessed the score range across age groups (3 to 85 years of age), test-retest reliability (intraclass correlation coefficient = 0.98), and concurrent and known-groups validity of grip strength.

When the final measures were recommended in all domains, the NIH Toolbox management core conducted a large national norming phase in 2011 to allow for normative comparison on each measure. All of the NIH Toolbox measures were administered to a total of 4859 participants, aged 3 to 85 years and representative of the US population. Hence, participants took the grip-strength test as part of the entire NIH Toolbox battery.

Participants were randomized into 4 different data-collection orders (ie, 4 study arms): cognition, motor, emotion, and sensation domains. Sampling was stratified by age, sex, and primary language (English, Spanish). Within each age stratum, target quotas were set according to the US population distribution of race, ethnicity, and level of education (parents' education for children). Preliminary results suggested no order effect for grip strength (P = .621). Detailed norming plans for the NIH Toolbox have been described.[2]

Participants

Grip-strength data collected in the cross-sectional normative phase of the NIH Toolbox project from August to November 2011 were analyzed. Inclusion required that an individual was (1) community dwelling and noninstitutionalized, (2) aged 3 to 85 years, (3) capable of following test instructions (English or Spanish), and (4) able to give informed consent or, in the case of children, to give assent with accompanying informed consent by proxy (ie, parent/guardian). Data were collected at 10 sites, primarily urban and suburban (Atlanta, Chicago-Oak Brook, Cincinnati, Columbus, Dallas, Los Angeles, Minneapolis, Philadelphia, Phoenix, and St Louis).

The norms of hand-grip strength for participants 3 to 17 years of age have been published.[8] Here, we only analyzed the adult data. From the initial 4859 data records, 3412 were removed because the age was younger than 18 years old. Subsequently, 91 were removed because of missing age or grip-strength data. An additional 124 were excluded as outliers because their grip-strength values were outside the 1.5 interquartile range of the mean for other participants of the same sex and age group, or because the between-side difference in grip strength was larger than 30%. **FIGURE 1** illustrates the data-cleaning process.

Data from 1232 remaining participants (aged between 18 and 85 years) were included in the final analysis. Of these, 449 (36.4%) were men and 783 (63.6%) were women. By self-report, 1151 (93.4%) participants in the sample were right-hand

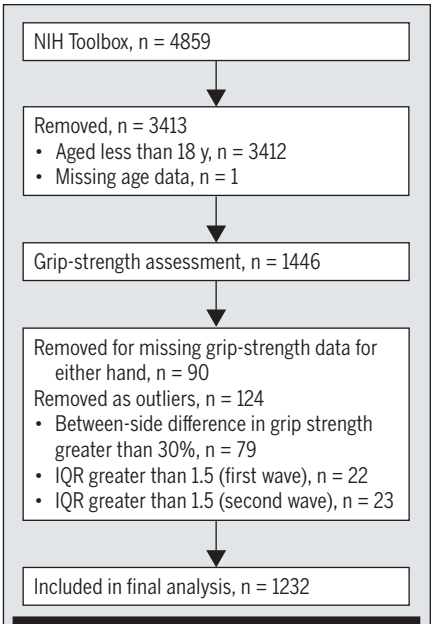

FIGURE 1. Data-cleaning flow chart. Abbreviations: IQR, interquartile range; NIH, National Institutes of Health.

dominant. The majority of the sample was white (77.8%, n = 958). Other races were represented as black or African American (12.5%, n = 154), American Indian or Alaska native (3.1%, n = 38), and Asian (2.6%, n = 32). Four hundred fifty-seven (37.1%) were Hispanic or Latino, while 774 (62.8%) were not. Regarding test instructions, 861 (69.9%) participants received the instructions in English, 276 (22.4%) in Spanish, and 95 (7.7%) in bilingual (English and Spanish) format.

The Institutional Review Board of Northwestern University approved the data-collection procedures. All participants consented to the study after being informed about the study's purpose and procedures. Additional Institutional Review Board approval was waived, as this was a secondary data analysis free of personal identifiers.

Grip-Strength Test Protocol

The test administrator's manual, which includes detailed descriptions of the protocol, and demonstration videos are available on the HealthMeasures website (http://www.healthmeasures.net) in both English (119 pages) and Spanish (156 pages).[36] Briefly, a calibrated digital Jamar dynamometer (Performance Health, Warrenville, IL), with its handle in the second position, was used. Participants were instructed to squeeze the dynamometer as hard as they could while seated in an upright posture, with arms by their sides, elbows flexed to 90°, and forearms in a neutral position.

The test administrator showed the dynamometer to the participant and said, "We will use this machine to measure how strong your hands are. You will squeeze the handle 2 times (1 practice and 1 test trial) with each hand while your arm is against your side and your elbow is bent like this (90°). The handle won't move, but the machine will show how hard you squeezed. See? (Show force measurement.) Do you have any questions?"

During the practice trial, the test administrator said, "Let's practice. First, we'll use this (dominant hand, your right or left) hand. When I say, 'Squeeze,' I want you to squeeze the handle hard, but not as hard as you can. [Support the dynamometer during testing.] Ready? 3, 2, 1, squeeze." After 3 to 4 seconds, the test administrator said, "Stop."

During the test trial, the test administrator said, "Now we're going to test each hand, beginning with this [Point and say, 'Your right or left'] hand. When I say, 'Squeeze,' this time I want you to squeeze the handle as hard as you can." The test administrator supported the dynamometer during testing.

Due to time constraints in the norming study of the entire NIH Toolbox battery, for grip-strength testing, a single submaximal practice trial was conducted for each hand, followed by a 30-second rest and a single maximal trial of 3 to 4 seconds for each hand. Participants were encouraged by the examiner, who chanted, "Harder, harder, harder." The force associated with the maximal trial was documented in pounds and later converted to kilograms.

Statistical Analysis

All analyses were performed using SPSS Statistics for Windows Version 23.0 (IBM Corporation, Armonk, NY). To compare hand-grip measures, participants were categorized into 13 age groups: 18 to 24, 25 to 29, 30 to 34, 35 to 39, 40 to 44, 45 to 49, 50 to 54, 55 to 59, 60 to 64, 65 to 69, 70 to 74, 75 to 79, and 80 to 85 years. A 2-by-2-by-13 (sex by side by age group) general linear model (GLM) analysis was used to test the hypothesis that these 3 independent variables should be used to stratify grip-strength measurements. For independent variables found to have a significant main or interactive effect on grip strength, pairwise post hoc comparisons were conducted using the GLM. The effect of other potentially explanatory variables on grip strength was also examined using the GLM. These variables included body mass index (BMI), ethnicity, education, language, and handedness. Descriptive statistics of grip strength were tabulated for relevant strata.

Prior to generating the regression equations, scatter plots and Pearson correlation coefficients were used to explore the relationships between grip strength and independent variables of interest, including height, weight, BMI, and age. Note that all 4 variables are continuous variables. Mukaka's[27] guideline was used to interpret correlation coefficients in medical research: greater than 0.9 (very high), 0.7 to 0.9 (high), 0.5 to 0.7 (moderate), 0.3 to 0.5 (low), and less than 0.3 (negligible). Data were checked for assumptions of normality, linearity, and multicollinearity. Four forward multiple linear regression analyses (1 for each sex and side category) were used to generate explanatory equations for grip strength.

The initial explanatory variables included age, age squared, age cubed, height, weight, and BMI. Age squared and age cubed were used because the scatter plots between grip strength and age showed a nonlinear relationship. Data points with Cook's distance greater than $4/(n-2)$, where n is the sample size, were considered as influential points[37] and were removed from the regression analysis. Model fit was inspected using the overall F test for the regression model, individual t tests for each regression coefficient, and adjusted R^2. Based on the numerous hypothesis tests conducted and a desire to reduce the risk of type I error, a significance level of $P<.005$ was adopted as an indicator of statistical significance.

RESULTS

FIGURE 2 illustrates the trajectories of grip-strength values from individuals 18 to 85 years of age. The age group is on the x-axis, while the mean grip strength in kilograms is on the y-axis and is presented by sex and side. General linear model results verified that grip strength differed significantly ($P<.001$) by sex (with men stronger than women), by side (with the dominant side stronger than the nondominant side), and by age group (with younger adults stronger than

older adults). No significant interaction effects were found. **TABLE 1** summarizes grip strength (kilograms) by sex, side, and age group. To assist in clinical interpretation of grip-strength data, the authors also reported the summary of hand-grip strength measures in pounds (**APPENDIX A**, available at www.jospt.org) and presented box plots of grip strength by sex and side in kilograms (**FIGURE 3**) and pounds (**APPENDIX B**, available at www.jospt.org).

Pairwise comparisons showed that grip strength for participants aged 18 to 24 years was not statistically different from participants in nearby age groups (25-59 years) but was significantly greater than that of participants in age groups of 60 years and older ($P<.001$). Further GLM analysis showed that Hispanic or Latino participants had lower grip strength than participants classified as not Hispanic or Latino ($P<.001$). Participants with a high school (HS) diploma (ie, HS graduate) and a higher degree (more than HS) were stronger than participants who did not finish secondary education (less than HS) ($P<.001$). Participants who were given instructions in English showed higher grip strength than participants who were given instructions in Spanish only ($P<.001$); however, the differences were not significant between bilingual (English and Spanish) and Spanish only ($P = .049$-$.720$). At a significance level of .005, grip-strength values were not statistically different by BMI category ($P = .045$) or by handedness ($P = .007$). **TABLE 2** summarizes these GLM comparisons.

Among demographic variables, correlations with grip strength were significant and moderate for height ($r = 0.61$) and significant and low for weight ($r =$

TABLE 1 Summary of Hand-Grip Strength Measurements by Side, Sex, and Age-Group Strata*

				Percentile				
Hand/Sex/Age, y	Height, m	Weight, kg	Strength, kg	10	25	50	75	90
Dominant								
Male								
18-24 (n = 36)	1.81 ± 0.08	82.0 ± 16.9	47.0 ± 8.1	36.2	41.4	47.8	51.2	57.9
25-29 (n = 35)	1.78 ± 0.07	86.4 ± 21.9	49.7 ± 11.6	33.7	43.3	49.3	59.4	66.2
30-34 (n = 29)	1.75 ± 0.06	91.0 ± 17.9	46.5 ± 12.1	31.2	36.4	46.1	56.4	63.1
35-39 (n = 41)	1.77 ± 0.07	92.5 ± 22.1	47.1 ± 11.9	30.3	39.7	50.1	54.3	60.8
40-44 (n = 47)	1.75 ± 0.07	90.0 ± 19.6	46.7 ± 11.7	34.3	39.9	45.9	54.4	63.1
45-49 (n = 32)	1.73 ± 0.06	89.1 ± 17.9	42.8 ± 10.9	31.1	35.8	40.7	48.2	59.2
50-54 (n = 46)	1.78 ± 0.08	93.8 ± 17.2	44.0 ± 10.3	30.4	39.0	44.8	52.3	56.7
55-59 (n = 27)	1.77 ± 0.08	92.3 ± 22.4	40.7 ± 10.4	28.2	32.4	38.7	47.8	56.3
60-64 (n = 33)	1.77 ± 0.08	90.3 ± 13.4	38.4 ± 10.3	23.3	30.4	40.3	44.9	52.5
65-69 (n = 22)	1.74 ± 0.08	86.2 ± 17.9	36.8 ± 10.5	17.8	31.5	36.6	45.8	50.1
70-74 (n = 39)	1.75 ± 0.08	88.3 ± 18.0	34.7 ± 9.0	16.7	29.3	36.3	41.2	45.6
75-79 (n = 24)	1.76 ± 0.08	86.2 ± 14.0	32.7 ± 10.1	18.4	25.9	33.5	36.6	43.5
80-85 (n = 38)	1.75 ± 0.08	81.1 ± 13.0	28.1 ± 9.1	15.6	21.5	29.5	34.6	38.2
Female								
18-24 (n = 54)	1.61 ± 0.07	72.3 ± 21.2	28.1 ± 7.1	17.6	22.4	28.4	33.8	38.0
25-29 (n = 102)	1.61 ± 0.07	73.3 ± 20.1	29.6 ± 7.0	20.2	25.4	29.6	33.6	39.7
30-34 (n = 109)	1.63 ± 0.07	76.1 ± 19.6	28.9 ± 6.2	20.5	23.9	29.8	33.0	37.1
35-39 (n = 90)	1.62 ± 0.07	75.2 ± 17.4	29.2 ± 6.2	20.0	24.5	30.3	33.0	38.0
40-44 (n = 88)	1.63 ± 0.07	75.9 ± 18.4	29.9 ± 6.2	22.8	26.5	30.4	33.8	37.4
45-49 (n = 52)	1.63 ± 0.08	79.7 ± 19.1	28.8 ± 7.2	17.7	25.2	28.7	34.4	37.6
50-54 (n = 65)	1.63 ± 0.07	75.6 ± 16.0	28.2 ± 6.3	19.7	24.6	28.2	32.7	35.2
55-59 (n = 30)	1.62 ± 0.07	76.6 ± 16.2	25.1 ± 6.2	16.9	20.7	24.1	30.2	32.2
60-64 (n = 58)	1.62 ± 0.07	76.7 ± 17.4	23.6 ± 6.5	15.9	19.2	24.4	28.1	31.8
65-69 (n = 29)	1.62 ± 0.07	80.0 ± 21.5	22.1 ± 6.6	11.7	19.3	22.2	25.0	31.2
70-74 (n = 43)	1.60 ± 0.07	77.4 ± 18.8	21.5 ± 5.1	15.2	19.5	22.5	23.9	27.5
75-79 (n = 17)	1.58 ± 0.08	66.7 ± 10.4	19.6 ± 6.0	12.6	15.7	18.2	22.4	27.8
80-85 (n = 46)	1.60 ± 0.06	70.0 ± 11.3	19.9 ± 4.4	14.5	16.6	19.5	21.8	27.0

Table continues on page 689.

0.34-0.36) and age (r = −0.26 to −0.27). In multiple regression analysis (forward selection), 89 participants with Cook's distance greater than 0.00325 (4/[1232 − 2]) were considered as influential points and were removed. Regression analysis resulted in age cubed, height, and weight being selected as the best explanatory variables, with adjusted R^2 values ranging from 0.32 to 0.44. The overall F test was significant (P<.001), as were the t tests for the 3 regression coefficients (P<.001). **TABLE 3** provides normative regression equations for describing grip strength. **APPENDIX C** (available at www.jospt.org) shows the scatter plots of the observed data and the predicted grip-strength values derived from the regression equations. Using the same existing data, the authors conducted a preliminary analysis examining the accuracy of the regression models. For men, mean ± SD differences between observed and predicted grip strength were −0.69 ± 9.6 and −0.62 ± 9.2 kg for dominant and nondominant hands, respectively. For women, mean ± SD differences between observed and predicted grip strength were −0.24 ± 6.1 and −0.30 ± 6.0 kg for dominant and nondominant hands, respectively.

DISCUSSION

This study presents normative reference values and equations for grip strength based on data obtained from the US population within the last 10 years. Notably, the data were acquired using procedures consistent with most published recommendations.[17,32] Normative data provided in this study may be beneficial for diverse medical and ergonomic research, enabling clinicians and researchers to compare the grip-strength values in individuals with or without impairments to the reference

TABLE 1 Summary of Hand-Grip Strength Measurements by Side, Sex, and Age-Group Strata* (continued)

				Percentile				
Hand/Sex/Age, y	Height, m	Weight, kg	Strength, kg	10	25	50	75	90
Nondominant								
Male								
18-24 (n = 36)	1.81 ± 0.08	82.0 ± 16.9	44.9 ± 7.8	35.7	38.0	44.5	50.4	55.2
25-29 (n = 35)	1.78 ± 0.07	86.4 ± 21.9	46.5 ± 9.6	31.1	39.4	47.2	56.4	59.9
30-34 (n = 29)	1.75 ± 0.06	91.0 ± 17.9	45.8 ± 11.3	28.5	37.0	45.0	56.2	60.1
35-39 (n = 41)	1.77 ± 0.07	92.5 ± 22.1	45.5 ± 11.0	34.3	37.5	47.2	52.3	58.8
40-44 (n = 47)	1.75 ± 0.07	90.0 ± 19.6	44.9 ± 11.7	32.1	38.8	42.7	52.4	61.4
45-49 (n = 32)	1.73 ± 0.06	89.1 ± 17.9	41.2 ± 10.0	29.6	34.4	40.4	46.5	57.7
50-54 (n = 46)	1.78 ± 0.08	93.8 ± 17.2	42.3 ± 10.6	27.1	38.3	44.3	48.7	55.1
55-59 (n = 27)	1.77 ± 0.08	92.3 ± 22.4	38.5 ± 9.6	27.4	30.7	37.2	42.5	55.3
60-64 (n = 33)	1.77 ± 0.08	90.3 ± 13.4	37.2 ± 9.1	23.4	31.9	37.1	44.8	49.3
65-69 (n = 22)	1.74 ± 0.08	86.2 ± 17.9	35.4 ± 10.3	17.3	28.0	37.5	43.0	48.0
70-74 (n = 39)	1.75 ± 0.08	88.3 ± 18.0	34.0 ± 9.5	20.5	29.9	34.5	40.6	45.7
75-79 (n = 24)	1.76 ± 0.08	86.2 ± 14.0	30.3 ± 9.9	14.5	24.5	30.2	36.0	40.2
80-85 (n = 38)	1.75 ± 0.08	81.1 ± 13.0	27.1 ± 9.4	14.2	20.0	27.3	32.0	40.0
Female								
18-24 (n = 54)	1.61 ± 0.07	72.3 ± 21.2	26.6 ± 6.4	20.0	21.8	24.7	31.0	37.6
25-29 (n = 102)	1.61 ± 0.07	73.3 ± 20.1	27.9 ± 6.6	20.4	23.7	27.5	31.8	38.2
30-34 (n = 109)	1.63 ± 0.07	76.1 ± 19.6	27.7 ± 5.9	19.6	24.1	27.6	30.8	35.2
35-39 (n = 90)	1.62 ± 0.07	75.2 ± 17.4	28.0 ± 6.0	19.7	23.7	27.6	32.0	36.4
40-44 (n = 88)	1.63 ± 0.07	75.9 ± 18.4	28.9 ± 6.4	21.7	25.2	29.3	33.6	36.8
45-49 (n = 52)	1.63 ± 0.08	79.7 ± 19.1	27.4 ± 7.0	17.1	22.8	26.9	33.4	36.5
50-54 (n = 65)	1.63 ± 0.07	75.6 ± 16.0	26.5 ± 6.5	17.7	22.3	26.4	31.9	34.8
55-59 (n = 30)	1.62 ± 0.07	76.6 ± 16.2	23.6 ± 6.4	14.6	18.4	23.5	28.2	31.1
60-64 (n = 58)	1.62 ± 0.07	76.7 ± 17.4	22.9 ± 6.3	15.8	17.6	22.6	28.2	30.6
65-69 (n = 29)	1.62 ± 0.07	80.0 ± 21.5	21.0 ± 6.6	15.0	16.2	21.4	25.8	30.6
70-74 (n = 43)	1.60 ± 0.07	77.4 ± 18.8	20.2 ± 5.5	13.7	16.7	20.9	23.5	28.0
75-79 (n = 17)	1.58 ± 0.08	66.7 ± 10.4	18.7 ± 5.8	10.7	14.4	18.6	22.0	27.4
80-85 (n = 46)	1.60 ± 0.06	70.0 ± 11.3	19.4 ± 4.0	13.9	17.3	19.3	21.0	24.5

Values are mean ± SD unless otherwise indicated.

TABLE 2 Comparisons of Hand-Grip Strength Measurements by Participant Characteristics*

		Men			Women	
Variable	**n**	**Dominant Hand**	**Nondominant Hand**	**n**	**Dominant Hand**	**Nondominant Hand**
BMI, kg/m^2						
<18.5 (underweight)	1	40.8	38.2	13	25.2 ± 3.7	25.1 ± 3.9
18.5-24.9 (normal weight)	125	39.9 ± 11.5	38.7 ± 10.7	236	27.0 ± 6.7	25.8 ± 6.4
25-29.9 (overweight)	161	42.2 ± 11.8	40.4 ± 11.6	238	27.5 ± 6.8	26.5 ± 6.8
≥30 (obese)	141	43.5 ± 12.9	41.7 ± 12.2	257	27.1 ± 8.0	25.6 ± 7.6
Ethnicity†						
Not Hispanic or Latino	288	43.6 ± 11.8	41.9 ± 11.1	486	27.8 ± 7.1	26.7 ± 7.0
Hispanic or Latino	160	38.0 ± 12.2	36.5 ± 11.9	297	25.9 ± 7.0	24.4 ± 6.6
Education†						
Less than high school	102	36.4 ± 13.0	34.6 ± 12.5	156	25.1 ± 7.3	24.1 ± 7.2
High school graduate	104	43.2 ± 11.8	41.3 ± 10.7	168	26.1 ± 6.9	25.0 ± 6.6
More than high school	213	44.5 ± 10.6	42.8 ± 10.3	409	28.3 ± 7.0	27.0 ± 6.8
Language of administration†						
English only	320	43.8 ± 11.6	42.0 ± 11.1	541	27.8 ± 7.1	26.6 ± 6.9
Spanish only	97	34.9 ± 12.5	33.8 ± 12.3	179	25.0 ± 6.9	23.3 ± 6.4
Bilingual, English primary	10	41.4 ± 10.7	41.0 ± 10.4	18	28.2 ± 5.7	26.5 ± 5.2
Bilingual, Spanish primary	22	39.9 ± 9.8	37.5 ± 8.4	45	26.8 ± 7.6	26.0 ± 7.2
Handedness						
Right handed	411	41.4 ± 12.4	39.7 ± 11.8	740	27.0 ± 7.1	25.7 ± 6.9
Left handed	38	43.9 ± 10.3	43.0 ± 10.4	43	28.5 ± 7.0	28.3 ± 7.0

Abbreviation: BMI, body mass index.
**Values are mean ± SD kilograms unless otherwise indicated.*
†The main effect was statistically significant (P<.001) for both dominant and nondominant hands. No main effect was statistically significant (P<.001) for 1 side only.

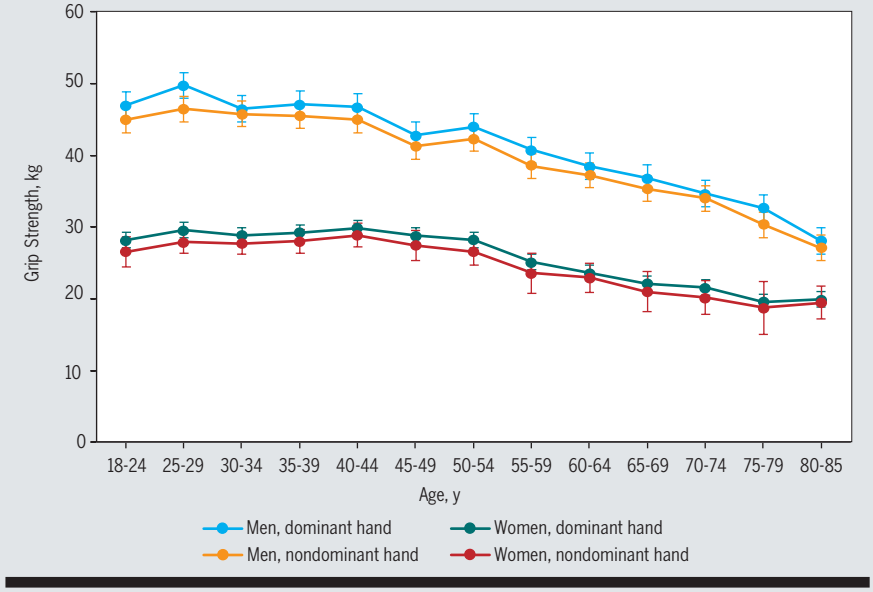

FIGURE 2. Trajectories of grip-strength measurements for individuals 18 to 85 years of age. The line indicates the average values of the hand-grip strength. The error bars indicate the 95% confidence interval for each age group.

values established based on the general, relatively healthy population. Clinicians and researchers could use grip strength to document progression of hand muscle strength and to provide feedback during the rehabilitation process with the knowledge of normal score ranges for individuals of similar age and sex.

Based on precedence and the results of this GLM analysis, the authors stratified normative reference values by side, sex, and age, with all of the age strata but 1 (18-24 years) limited to 5-year spans. In addition to means and standard deviations for each stratum, the authors provided percentiles of grip strength to assist clinicians and researchers in interpreting measurements obtained from tested individuals. The values presented are mostly less than, but within 1 standard deviation of, those provided by Dodds et al[14]

for participants in 12 British studies. This difference may be due to the use of maximum grip strength of the participants in the Dodds et al[14] study, whereas the present study used the results of 1 maximum effort for each hand. However, it may be that the grip strength of the 2 populations is different. The values presented are also less than, but usually within the confidence intervals of, those derived by Bohannon et al[6] through meta-analysis. Those values, however, were presented for the right and left sides rather than for the dominant and nondominant sides, as in this study.

All of the data consolidated by Bohannon et al[6] and most of the data consolidated by Dodds et al[14] were obtained before the data of the NIH Toolbox normative phase study. Previously collected grip-strength reference values may not be the best standard by which to interpret the performance of individuals tested more recently. This was highlighted in a study by Fain and Weatherford[16] that compared the grip strength of young adults (millennials) with the normative reference values obtained over 30 years prior by Mathiowetz et al[25] and found the more recently tested individuals to be weaker.

Although reference values stratified according to significant explanatory variables are useful, they may not provide as specific a referent as a regression equation. Reference equations for grip strength have been reported before and tend to include age, sex, height, and weight as independent variables.[15] Based on correlational and regression analysis, the authors of this study identified 3 variables for inclusion in reference equations (weight, height, and age cubed). Of these, only age was used to stratify reference values. Sex and side, which are not included in the regression equations, are addressed by providing separate equations for each sex and side stratum. Notably, BMI was not included in this study's equations. Although grip-strength values are sometimes interpreted in light of BMI,[18] grip strength was found in this

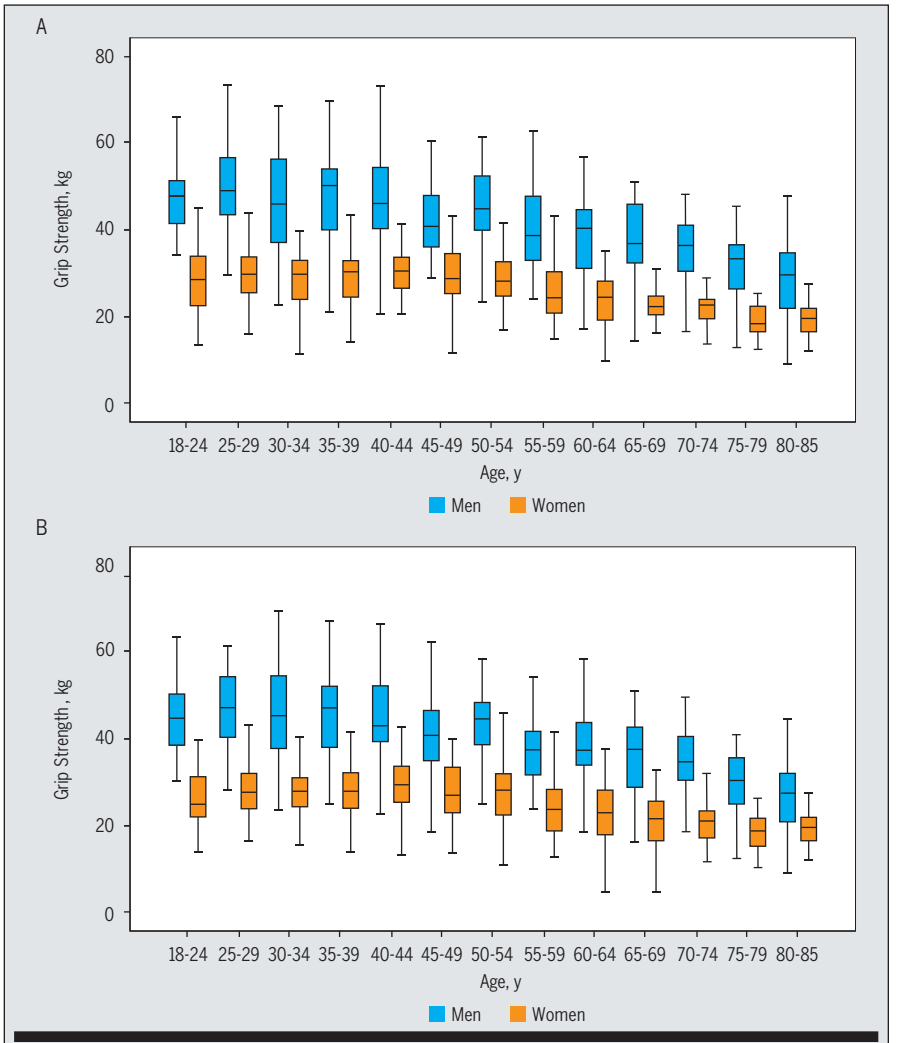

FIGURE 3. Box plot of the hand-grip strength measurements (kilograms) for (A) the dominant hand and (B) the nondominant hand. For each box, the center line represents the median (50th percentile), the height of the box represents interquartile range (25th percentile and 75th percentile), and the top error bars represent minimum and maximum (after removing outliers).

TABLE 3 Reference Equations of Grip Strength by Sex and Hand Dominance

Sex/Hand Dominance	Reference Equation	Adjusted R^2
Male		
Dominant	$-29.959 - 3.095E^{-05} \times (age^3) + 38.719 \times (height) + 0.113 \times (weight)$	0.44
Nondominant	$-30.474 - 2.937E^{-05} \times (age^3) + 38.594 \times (height) + 0.099 \times (weight)$	0.44
Female		
Dominant	$-22.717 - 1.920E^{-05} \times (age^3) + 30.360 \times (height) + 0.048 \times (weight)$	0.36
Nondominant	$-21.292 - 1.776E^{-05} \times (age^3) + 28.995 \times (height) + 0.040 \times (weight)$	0.32

**Age in years, height in meters, and weight in kilograms. E is the scientific notation. E^{-05} indicates $10^{-5} = 0.00001$. For example, the predicted grip strength of the dominant hand for a 42-year-old man with a height of 1.7 m and weight of 67.1 kg is $-29.959 - 3.095 \times 10^{-5} \times (42^3) + 38.719 \times (1.7) + 0.113 \times (67.1) = 41.2$ kg.*

study to be correlated more highly with height than with BMI. Once the correlations of height and weight with grip strength were controlled, BMI made no further contribution to the explanation of grip strength.

A strength of this study is that it was based on the secondary analysis of data from a population-based study with rigorous sampling plans, data-collection procedures, and quality-control practices. An NIH Toolbox administration manual was provided for all sites to help with test standardization. In addition, examiners participating in the project were trained on standardized procedures prior to data collection. The authors of this study presented norms of hand-grip strength in the general population, following the inclusion criteria listed by the NIH Toolbox. Norms are statistics that describe the test performance of a predefined population (community-dwelling, noninstitutionalized adults in the present study). As normative data are not exactly the same as unaffected data, the data set may include individuals with comorbidities. Previous findings supported the 10% rule, which states that the dominant hand possesses a 10% greater grip strength than the nondominant hand in healthy subjects, and the differences are more noticeable for right-handed individuals.[29] Hence, as a conservative estimate, the researchers excluded 6.4% (n = 79) of grip-strength data, in which the between-side difference in grip strength was larger than 30%. Note that there were 54.3%, 84.9%, and 94.6% of data with side differences less than or equal to 10%, 20%, and 30%, respectively. The absence of data may reduce statistical power and the representativeness of the samples, and may cause bias in the analysis. However, side differences greater than 30% may indicate entry error or uncertain pathology. Because it is not feasible to check the primary source, the authors believe that removing side differences greater than 30% will improve the representation of the relatively healthy population, which facilitates the data interpretation. Additionally, inspecting the side differences provided a way to check potential data-entry errors, as the authors did find subjects to have extreme hand-grip values of 105 kg in the dominant hand, but 1 kg in the nondominant hand.

Several limitations of the study must also be acknowledged. First, hand grip was measured as part of the entire set of NIH Toolbox measures. It is uncertain whether this may have affected the forces obtained. Second, although the sample was relatively large, its size was diminished by missing values (eg, demographics) and the exclusion of extreme values (outliers, data-entry errors). Ultimately, the sample was too small to justify stratification for factors (eg, ethnicity) determined to be relevant to grip strength.

Third, as this is a secondary data analysis, researchers were not in control of the data-collection procedures, and variables that may be important to grip strength (eg, hand size) were not addressed. Indeed, they could not be, as they were not included among the NIH Toolbox measures. Similarly, the authors do not have information about the number of examiners/raters and the inherent interrater reliability during the norming phase. Fourth, the researchers applied multiple regression analysis using the 4 independent variables that were available. To increase the predictive power of grip strength across the age span, more relevant variables (eg, hand size, ethnicity, comorbid burden, occupation) and more complicated statistical methods (such as Box-Cox power transformation and nonlinear regression models) could be explored. Last, future studies should endeavor to validate normative regression equations using a new and independent sample.

CONCLUSION

The normative reference values provided herein can serve as a guide for interpreting grip-strength measurements obtained from adults in the United States. Reference equations can be used to the same end. ◉

KEY POINTS

FINDINGS: This project provides US population-based, grip-strength reference values obtained from the US National Institutes of Health Toolbox project. These hand-grip values are summarized after stratification by side (ie, dominant and nondominant), sex, and age. Reference equations are provided.

IMPLICATIONS: The reference values provided can be used to interpret grip-strength measures obtained from adults in the United States.

CAUTION: The reference values and equations provided were obtained from a sample of insufficient size to justify additional stratification by race and other such factors.

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APPENDIX A

SUMMARY OF HAND-GRIP STRENGTH MEASUREMENTS BY SIDE, SEX, AND AGE-GROUP STRATA

		Percentile				
Hand/Sex/Age, y	**Strength, lb***	**10**	**25**	**50**	**75**	**90**
Dominant						
Male						
18-24 (n = 36)	103.6 ± 17.9	79.8	91.3	105.4	112.9	127.6
25-29 (n = 35)	109.6 ± 25.6	74.3	95.5	108.7	131.0	145.9
30-34 (n = 29)	102.5 ± 26.7	68.8	80.2	101.6	124.3	139.1
35-39 (n = 41)	103.8 ± 26.2	66.8	87.5	110.5	119.7	134.0
40-44 (n = 47)	103.0 ± 25.8	75.6	88.0	101.2	119.9	139.1
45-49 (n = 32)	94.4 ± 24.0	68.6	78.9	89.7	106.3	130.5
50-54 (n = 46)	97.0 ± 22.7	67.0	86.0	98.8	115.3	125.0
55-59 (n = 27)	89.7 ± 22.9	62.2	71.4	85.3	105.4	124.1
60-64 (n = 33)	84.7 ± 22.7	51.4	67.0	88.8	99.0	115.7
65-69 (n = 22)	81.1 ± 23.1	39.2	69.4	80.7	101.0	110.5
70-74 (n = 39)	76.5 ± 19.8	36.8	64.6	80.0	90.8	100.5
75-79 (n = 24)	72.1 ± 22.3	40.6	57.1	73.9	80.7	95.9
80-85 (n = 38)	61.9 ± 20.1	34.4	47.4	65.0	76.3	84.2
Female						
18-24 (n = 54)	61.9 ± 15.7	38.8	49.4	62.6	74.5	83.8
25-29 (n = 102)	65.3 ± 15.4	44.5	56.0	65.3	74.1	87.5
30-34 (n = 109)	63.7 ± 13.7	45.2	52.7	65.7	72.8	81.8
35-39 (n = 90)	64.4 ± 13.7	44.1	54.0	66.8	72.8	83.8
40-44 (n = 88)	65.9 ± 13.7	50.3	58.4	67.0	74.5	82.5
45-49 (n = 52)	63.5 ± 15.9	39.0	55.6	63.3	75.8	82.9
50-54 (n = 65)	62.2 ± 13.9	43.4	54.2	62.2	72.1	77.6
55-59 (n = 30)	55.3 ± 13.7	37.3	45.6	53.1	66.6	71.0
60-64 (n = 58)	52.0 ± 14.3	35.1	42.3	53.8	61.9	70.1
65-69 (n = 29)	48.7 ± 14.6	25.8	42.5	48.9	55.1	68.8
70-74 (n = 43)	47.4 ± 11.2	33.5	43.0	49.6	52.7	60.6
75-79 (n = 17)	43.2 ± 13.2	27.8	34.6	40.1	49.4	61.3
80-85 (n = 46)	43.9 ± 9.7	32.0	36.6	43.0	48.1	59.5
Nondominant						
Male						
18-24 (n = 36)	99.0 ± 17.2	78.7	83.8	98.1	111.1	121.7
25-29 (n = 35)	102.5 ± 21.2	68.6	86.9	104.1	124.3	132.1
30-34 (n = 29)	101.0 ± 24.9	62.8	81.6	99.2	123.9	132.5
35-39 (n = 41)	100.3 ± 24.3	75.6	82.7	104.1	115.3	129.6
40-44 (n = 47)	99.0 ± 25.8	70.8	85.5	94.1	115.5	135.4
45-49 (n = 32)	90.8 ± 22.0	65.3	75.8	89.1	102.5	127.2
50-54 (n = 46)	93.3 ± 23.4	59.7	84.4	97.7	107.4	121.5
55-59 (n = 27)	84.9 ± 21.2	60.4	67.7	82.0	93.7	121.9
60-64 (n = 33)	82.0 ± 20.1	51.6	70.3	81.8	98.8	108.7
65-69 (n = 22)	78.0 ± 22.7	38.1	61.7	82.7	94.8	105.8
70-74 (n = 39)	75.0 ± 20.9	45.2	65.9	76.1	89.5	100.8
75-79 (n = 24)	66.8 ± 21.8	32.0	54.0	66.6	79.4	88.6
80-85 (n = 38)	59.7 ± 20.7	31.3	44.1	60.2	70.5	88.2

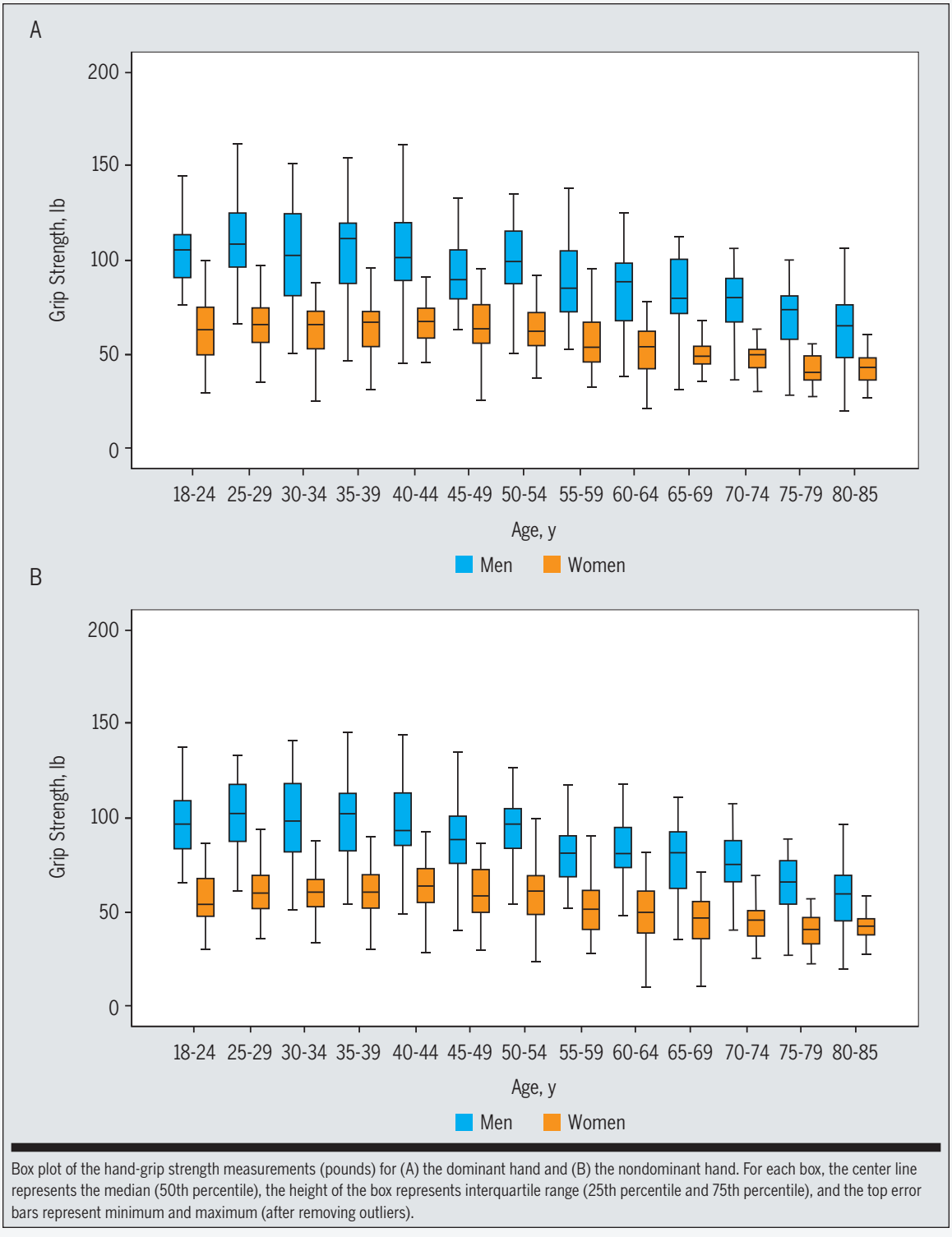
Table continues on page B2.

APPENDIX A

		Percentile				
Hand/Sex/Age, y	**Strength, lb***	**10**	**25**	**50**	**75**	**90**
Female						
18-24 (n = 54)	58.6 ± 14.1	44.1	48.1	54.5	68.3	82.9
25-29 (n = 102)	61.5 ± 14.6	45.0	52.2	60.6	70.1	84.2
30-34 (n = 109)	61.1 ± 13.0	43.2	53.1	60.8	67.9	77.6
35-39 (n = 90)	61.7 ± 13.2	43.4	52.2	60.8	70.5	80.2
40-44 (n = 88)	63.7 ± 14.1	47.8	55.6	64.6	74.1	81.1
45-49 (n = 52)	60.4 ± 15.4	37.7	50.3	59.3	73.6	80.5
50-54 (n = 65)	58.4 ± 14.3	39.0	49.2	58.2	70.3	76.7
55-59 (n = 30)	52.0 ± 14.1	32.2	40.6	51.8	62.2	68.6
60-64 (n = 58)	50.5 ± 13.9	34.8	38.8	49.8	62.2	67.5
65-69 (n = 29)	46.3 ± 14.6	33.1	35.7	47.2	56.9	67.5
70-74 (n = 43)	44.5 ± 12.1	30.2	36.8	46.1	51.8	61.7
75-79 (n = 17)	41.2 ± 12.8	23.6	31.7	41.0	48.5	60.4
80-85 (n = 46)	42.8 ± 8.8	30.6	38.1	42.5	46.3	54.0

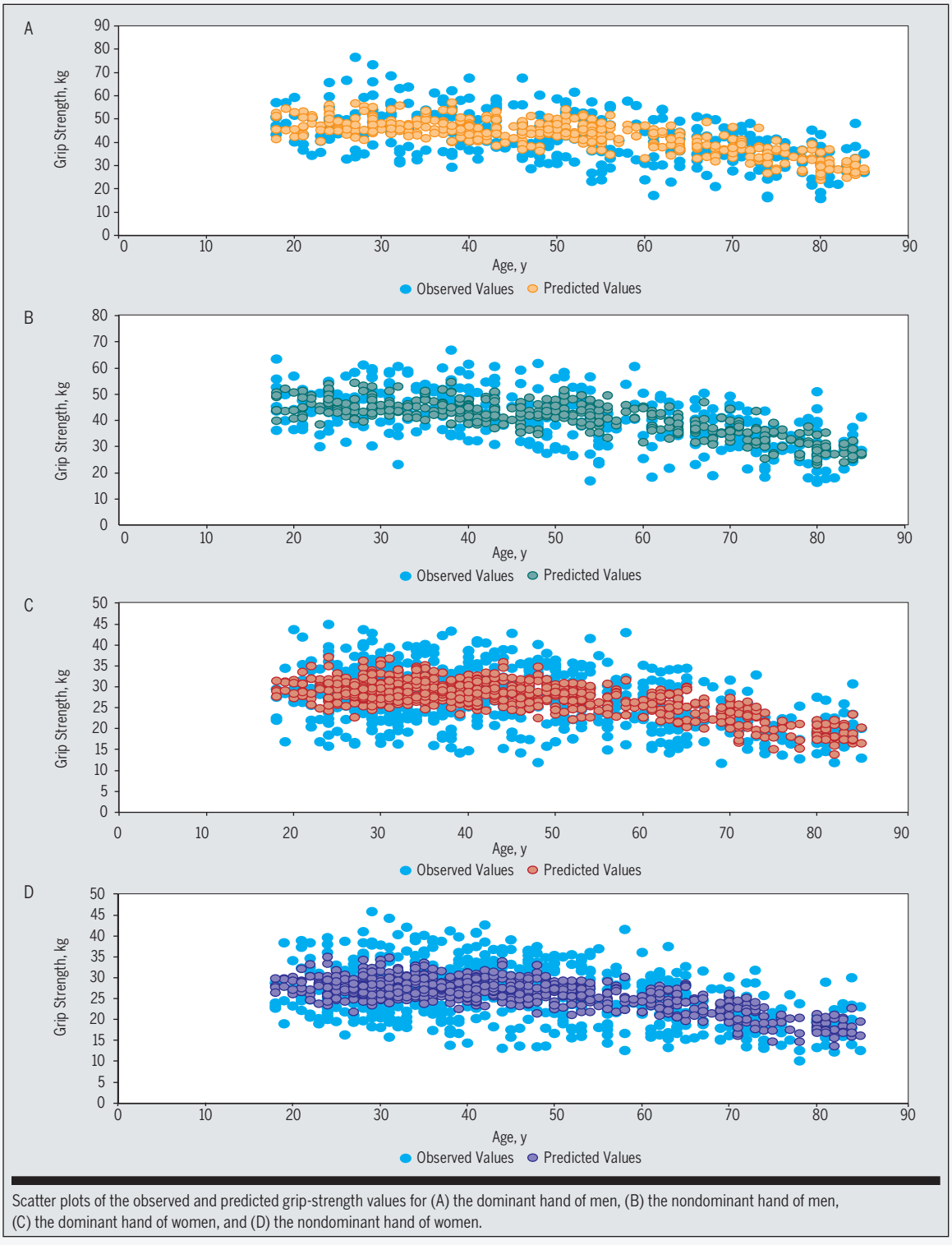
**Values are mean ± SD.*

APPENDIX B



Box plot of the hand-grip strength measurements (pounds) for (A) the dominant hand and (B) the nondominant hand. For each box, the center line represents the median (50th percentile), the height of the box represents interquartile range (25th percentile and 75th percentile), and the top error bars represent minimum and maximum (after removing outliers).

APPENDIX C



Scatter plots of the observed and predicted grip-strength values for (A) the dominant hand of men, (B) the nondominant hand of men, (C) the dominant hand of women, and (D) the nondominant hand of women.