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Tibiofemoral Osteoarthritis and Varus-Valgus Laxity

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Abstract

The purpose of this study was to systematically review and synthesize the literature measuring varus-valgus laxity in individuals with tibiofemoral osteoarthritis (OA). Specifically, we aimed to identify varus-valgus laxity differences between persons with OA and controls, by radiographic disease severity, by frontal plane knee alignment, and by sex. We also aimed to identify if there was a relationship between varus-valgus laxity and clinical performance and self-reported function. We systematically searched for peer-reviewed original research articles in PubMed, Scopus, and CINAHL to identify all existing literature regarding knee OA and objective measurement of varus-valgus laxity in vivo. Forty articles were identified that met the inclusion criteria and data were extracted. Varus-valgus laxity was significantly greater in individuals with OA compared with controls in a majority of studies, while no study found laxity to be significantly greater in controls. Varus-valgus laxity of the knee was reported in persons with OA and varying degrees of frontal plane alignment, disease severity, clinical performance, and self-reported function but no consensus finding could be identified. Females with knee OA appear to have more varus-valgus laxity than males. Meta-analysis was not possible due to the heterogeneity of the subject populations and differences in laxity measurement devices, applied loading, and laxity definitions. Increased varus-valgus laxity is a characteristic of knee joints with OA. Large variances exist in reported varus-valgus laxity and may be due to differences in measurement devices. Prospective studies on joint laxity are needed to identify if increased varus-valgus laxity is a causative factor in OA incidence and progression.

Keywords

osteoarthritis; knee; tibiofemoral; laxity; frontal plane

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Conflict of Interest

None of the authors have any competing interests that could potentially or inappropriately influence this work or its conclusions.

Osteoarthritis (OA) is the leading cause of disability in the United States, affecting more than 50 million adults.¹ OA is a multifactorial disease, with the mechanical environment of the joint playing a key role in cartilage synthesis and degeneration. Cartilage thickness varies by compartment in the knee joint^{2, 3} and, in healthy knees, increased cartilage thickness is seen on the regions experiencing the largest loads.^{4, 5} However, in patients with OA, increased loading is associated with decreased cartilage thickness and worsening disease severity.⁵⁻⁸ This paradoxical response to loading may be due to changes in the mechanical environment of the knee, such as altered joint laxity. Altered laxity may change knee kinematics and shift cartilage contact to areas that are not well conditioned, leading to a cycle of cartilage degeneration with increased loading (Fig. 1).^{5, 9-11}

To investigate the potential link between laxity and OA, several techniques have been used to assess knee joint laxity. Clinically, knee laxity is graded during a physical exam by manually applying a force to the tibia while stabilizing the femur in an unloaded position. Manual testing is subjective by nature, and inter- and intratester reliability of clinical varus-valgus instability in patients with knee OA has been shown to be poor ($\kappa = 0.23$ and $\kappa = 0.55$, respectively).¹² Consequently, a variety of devices have been developed in an attempt to quantify laxity with greater accuracy and precision, since such accuracy and precision would allow for the statistical testing of hypothetical quantitative links between knee laxity and OA. Tallroth and Lindholm first measured in vivo varus-valgus laxity in osteoarthritic knees utilizing stress radiographs.¹³ A device applied a varus or valgus load to the knee and the change in medial and lateral joint space widths was recorded. Brage et al measured angular rotation of the tibia with respect to the femur with a goniometer while varus and valgus loading was applied.¹⁴ Multiple other frontal plane knee laxity devices have subsequently been developed to quantify varus-valgus knee laxity by measuring a change in joint space or angular rotation with applied load.

The purpose of this study was to assess the relationship between varus-valgus laxity and tibiofemoral OA, by systematically reviewing and synthesizing the available literature. Specifically, we aimed to identify varus-valgus laxity differences between each of the following: subjects with OA and controls; by radiographic disease severity; by frontal plane knee alignment; by sex; and by clinical performance and self-reported function. We focused on varus-valgus laxity because it is commonly assessed clinically and multiple research groups have developed and published work regarding frontal plane laxity.

Materials and Methods

Search Terminology

We executed a systematic search of peer-reviewed research articles in the search engines PubMed, Scopus, and CINAHL published between January 1, 1966 and December 17, 2015. Our intent was to identify all existing literature regarding knee OA and varus-valgus laxity. The search strategy used three combined terms to identify articles with “knee ‘or’ tibiofemoral,” “osteoarthritis ‘or’ OA, ‘or’ degenerative joint disease ‘or’ joint degeneration,” and “stability ‘or’ instability ‘or’ laxity.” The results from these three searches were combined with ‘and’ statements and further filtered for articles in English with human subjects.

Study Selection

The inclusion criteria for studies in this systematic review were:

- Peer-reviewed, primary research articles published in English between January 1, 1966 and December 17, 2015.
- Human subjects.
- Confirmed radiographic tibiofemoral OA.
- Measurement of varus-valgus displacement on a continuous scale.

The exclusion criteria were:

- Cadaver studies.
- Case reports, conference abstracts, dissertations/theses.
- Review articles.
- Subjective, clinical assessment of varus-valgus laxity.
- Laxity only measured following a bone, ligament, or meniscal alteration made in the operating room to assess the surgical knee procedure (e.g., osteotomy or arthroplasty)

Data Extraction

The following information was extracted from each selected article if available: total number of subjects with OA, total number of knees investigated, number of male and female subjects, age, radiographic severity of OA, laxity testing method, quantified varus-valgus laxity, total number of controls, and load applied and knee flexion angle during testing.

Quality Assessment

Quality assessments were completed on the selected articles utilizing the study quality assessment tools developed for a systematic evidence review by the National Institutes of Health (NIH) Lifestyle Work Group.¹⁵ There is no clear consensus on the quality assessment method to use for systematic reviews of observation studies,¹⁶ however, the NIH Lifestyle Work Group tools had separate checklists and instructions for observational cohort/cross-sectional studies and case-control designs. This provided an additional level of detail in our systematic evidence review, and was the determining factor in choosing this method.

Results

The systematic review flow diagram outlining selection and exclusion is shown in Fig. 2 per Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.¹⁷ Exclusion criteria were applied in the order listed on Fig. 2. Abstracts from 2,385 articles were reviewed following the database search. The full texts of 131 articles were further examined and 40 articles met the inclusion. The reference lists of the final set of articles were checked to identify additional manuscripts to include in the review, but every article of interest had already been included in the initial search. Of the 40 selected studies, 4

were prospective trials,^{18–21} 16 were cohort-control designs,^{11, 14, 22–35} and the remaining 20 were cross-sectional studies.^{13, 36–54} Laxity was a primary variable of interest in 32 of the selected studies. Two studies received a quality assessment of poor with respect to the reporting of varus-valgus laxity in subjects with OA^{23, 24} due to a lack of detail on the method of objective laxity measurement.

The selected studies were categorized by topic with respect to OA and varus-valgus laxity for this systematic review: OA versus controls ($N=17$), radiographic OA severity ($N=7$), frontal plane knee alignment ($N=4$), sex differences ($N=1$), and clinical and self-reported measures of function ($N=18$). Studies could be included in multiple categories if analysis was completed on each topic. Ten studies did not fit into the categories above but quantified varus-valgus knee laxity in subjects with OA. The extracted data for these studies can be found in Supplementary Table S1 (online-only).

Varus-valgus laxity was quantified utilizing 11 different testing devices. These studies measured total varus-valgus laxity ($N=21$), separate varus or valgus laxity ($N=6$), specific medial and lateral compartment joint space ($N=11$), or combined joint space translation ($N=2$). The units of measure were degrees of varus/valgus angular excursion or mm of joint space. Some studies applied a force directly to the knee ($N=13$) while others applied the load away from the knee joint center to create a moment ($N=25$). With the devices that applied load directly to the knee, the femur and tibia were braced away from the joint center to create three-point bending. The distance between the reaction loads and the joint center creates a moment in the frontal plane which allowed for varus-valgus laxity assessment. Load application method was not reported in two selected studies and the magnitudes of applied loading were variable between articles. Laxity was measured at 20 degrees of knee flexion for the majority of studies ($N=32$), however, four studies used various knee flexion angles ranging from maximum extension to maximum flexion, and four studies did not report knee flexion angle. Several of the devices are listed in Tables 1 and 2, with the details for each study found in Supplementary Table S1 (online-only).

Laxity definitions were not consistent between studies and, in some cases, it was not possible to identify when identical subjects were included in subsequent manuscripts from the same research group. Study populations were also not homogeneous by severity of radiographic OA and predominant compartment affected by the disease. Furthermore, the devices used to quantify laxity varied in varus or valgus motion measured and load applied during testing. Due to these inconsistencies, a meta-analysis was not possible. A qualitative overview of findings for each topic can be seen in Table 3.

Osteoarthritis versus Controls

Seventeen studies included varus-valgus laxity of a control group. Of these studies, 11 found a significant increase in some measure of laxity in the OA group compared with controls,^{11, 23, 25–31, 33, 34} 4 found no difference,^{14, 22, 32, 35} and 2 did not report any statistical comparisons between groups as this was not the focus of those manuscripts.^{20, 24} In the studies which found increased laxity, four reported increased total varus-valgus laxity,^{11, 23, 25, 31} six had increased medial, but not lateral, compartment laxity in subjects with medial knee OA,^{26–30, 34} and one found increased total varus-valgus laxity after stair

climbing but not before activity.³³ Quantified varus-valgus angular laxity and joint space laxity results from these 17 studies are shown in Table 1 and Table 2, respectively.

Radiographic Osteoarthritis Severity

Varus-valgus laxity stratified by severity level of OA was reported in seven articles. Three studies found a significant difference between OA severity levels,^{11, 14, 38} while four found no difference in laxity by severity level.^{13, 22, 32, 33} In the studies that found a significant result, Brage et al found knees with mild OA to have more laxity than moderate or severe OA groups, 15 (4.8) versus 10.9 (3.9) and 10.4 (3.6) degrees respectively.¹⁴ In this study, mild OA was indicated by the presence of osteophytes but less than 50% loss of joint space. Sharma et al found the opposite relationship between OA severity and laxity. Patients with Kellgren-Lawrence (KL) grade 2 exhibited significantly less laxity than subjects with KL grade 3 or 4, 4.4 (0.16) versus 5.1 (0.22) and 5.7 (0.30) degrees, respectively.¹¹ van der Esch et al reported knees with a small amount of joint space narrowing to have more laxity than knees without joint space narrowing.³⁸

Frontal Plane Knee Alignment

Four studies measured laxity categorized by frontal plane alignment. Two found significant differences between aligned/malaligned and least/most varus aligned groups,^{38, 43} while the other two studies did not run any statistical tests comparing alignment groups.^{20, 47} van der Esch et al found that malaligned knees, characterized by greater than 5 degrees of mechanical varus or valgus alignment, had significantly increased total laxity compared with aligned knees, 9.4 (3.9) versus 6.6 (3.9) degrees, respectively.³⁸ However, no statistical difference was found between the total laxity of the varus malaligned group and the valgus malaligned group.³⁸

Lim et al reported that the most varus aligned group had significantly less varus laxity when compared with the group with the least varus alignment, 4.9 (2.0) versus 6.5 (2.5) degrees.⁴³ Eriksson et al only tested medial joint space changes in varus knees and lateral joint space changes for valgus knees, thus a comparison could not be made.⁴⁷ Sharma et al reported similar total laxities for subjects with neutral, varus, and valgus alignments, 4.1 (2.6), 3.9 (2.5), and 3.8 (2.7) degrees, respectively, but did not test for any difference between alignment groups.²⁰

Sex Difference

Only one study reported the laxity of males and females with OA separately. van der Esch et al found females to have significantly more varus-valgus laxity than males, 7.7 (2.9) versus 4.6 (2.2) degrees, respectively.⁴¹ Another study selected in the systematic review found women to have larger varus-valgus laxity than men in healthy controls.¹¹

Clinical and Self-reported Function

Varus-valgus laxity and a measure of clinical or self-reported function were reported in 18 studies.^{18, 19, 24, 25, 28–30, 35, 37, 39, 43–46, 48–50, 53} Of these studies, only seven tested for a relationship between varus-valgus laxity and function. Sharma et al found that greater laxity was associated with a weaker relationship between low extremity strength and physical

function.³⁷ Physical function was characterized in this study by chair-stand rate and self-reported activity limitations, which was quantified by the Western Ontario and McMaster Universities Arthritis Index physical function subscale (WOMAC-PF). This study also found that greater varus-valgus laxity was modestly associated with worse WOMAC-PF score, but there was no significant association with laxity and chair-stand rate. In a subsequent study which tested over a 3-year period, Sharma et al found increased varus-valgus laxity significantly increased the likelihood of a poor WOMAC-PF outcome, however, varus-valgus laxity was not related to chair-stand rate.¹⁸ van der Esch et al found increased varus-valgus laxity to be moderately associated with faster 100 m walking time and decreased lower extremity strength,³⁹ yet no direct association was found between varus-valgus laxity and WOMAC-PF. Laxity and muscle strength both significantly explained the variance in WOMAC-PF in a multivariate regression, with increased laxity and strength improving WOMAC-PF. In a separate multivariate regression, varus-valgus laxity did not contribute to 100 m walking time, but the interaction between varus-valgus laxity and muscle strength significantly reduced time needed to complete task. Holla et al found increased laxity to be significantly associated with less activity limitations (WOMAC-PF) but not significantly associated with the timed stair-climbing test, in a multivariate regression including strength.⁴⁸ In two separate studies, Schmitt et al found no significant differences in varus or valgus laxity between groups separated by self-reported perception of instability.^{44, 45} Knoop et al, Gustafson et al, and Chang et al also found high varus-valgus joint laxity was not associated with self-reported perception of instability.^{35, 49, 53}

Discussion

The purpose of this study was to systematically review and synthesize the literature measuring varus-valgus laxity in subjects with tibiofemoral OA. Varus-valgus laxity has been hypothesized to influence cartilage health, but given the variance in reported laxity, it is difficult to draw conclusions from the literature as a whole. Specifically, we aimed to identify varus-valgus laxity differences between subjects with OA and controls, by radiographic disease severity, by frontal plane knee alignment, by sex, and if there was a relationship to clinical performance and self-reported function. Reported varus-valgus laxity varied greatly between studies using the 11 separate testing devices, and it is very difficult to compare studies measuring joint space change to those quantifying angular rotation. Large variations were also found when comparing similar devices measuring angular rotation. Paradoxically, total varus-valgus laxity was often smaller using devices which applied larger loads about the knee joint. These results may be due to differences between testing devices or differences in subject populations. Variance in OA severity between studies is likely not the cause of this paradoxical relationship, since this phenomenon of increased laxity with less applied load still exists between similar levels of OA, as demonstrated in Table 1. The following considerations have been previously identified as necessary to improve laxity measurement and reduce variation compared with the clinical examination: measure at a consistent knee flexion angle, reduce artifact associated with estimating bone position from measurements made over soft tissue, reduce muscular guarding during manipulation, and accurately measure the applied load and the tibiofemoral motion.^{11, 12, 55, 56} Regarding these considerations, there do not appear to be drastic differences or shortcomings between testing

devices that could explain the reported variations. A “gold standard” of laxity measurement is necessary to confirm the accuracy and precision of the varied testing devices. This would potentially allow for meta-analyses in the future, using validated testing devices.

Subjects with OA exhibited increased frontal plane laxity compared with controls. Across the comparisons made in the 40 systematically selected studies, this finding was the most consistent. The majority of studies were not designed in a manner to distinguish if altered varus-valgus laxity leads to OA, but altered laxity was present in subjects with OA. This finding lends support to the theory that altered varus-valgus laxity is a component of OA and more research is needed to identify if cartilage degeneration is due to a change in joint contact patterns resulting from altered laxity.^{5, 9}

In the seven articles that reported laxity based on severity of OA, the results and conclusions were conflicted. Brage et al and van der Esch et al found that knees with more severe radiographic OA exhibited less laxity,^{14, 38} while Sharma et al found increasing laxity with increasing disease severity, after an initial decrease in laxity from KL grade 0/1 to KL grade 2.¹¹ The formation of osteophytes tend to stabilize the knee^{36, 42} and radiographic scales use combinations of osteophyte formation, joint space narrowing, and bone deformity to characterize severity of OA. The increased laxity seen in subjects with early radiographic signs of OA indicates that osteoarthritic changes may worsen after an initial increase in laxity, in an effort to control excessive knee joint motion. Increased muscular cocontraction may be a strategy used to stabilize a knee joint,^{45, 57} thus increasing load on cartilage and potentially increasing the rate of degeneration.

In subjects with severe OA, laxity is hypothesized to increase due to cartilage degeneration and bone erosion. This may create “pseudolaxity” by reducing the distance between ligament attachment points^{26, 58} and lead to greater measured laxity in subjects with severe OA. However, the trend of increased laxity in subjects with severe OA was only found in one of the three studies. The findings identified in this systematic review are not consistent and additional research is necessary to clarify the relationship between laxity and OA severity.

Static alignment changes may result from combined bone erosion and ligament adaptation, which could lead to unbalanced laxity in the varus and valgus directions. The two studies that statistically tested for differences in laxity by alignment found conflicting results.^{38, 43} These studies did not use the same definitions for malalignment and laxity measurement was varied, so it is difficult to compare results and form a consensus. Lim et al reported that the most varus aligned group had significantly less varus laxity when compared with the group with the least varus alignment,⁴³ while van der Esch found no statistical difference between the total laxity of the varus malaligned group and the valgus malaligned group.³⁸ The separation of varus and valgus laxity may be necessary when including subjects with predominantly medial or lateral knee OA in the same analysis. Alignment changes are often accompanied by increased disease severity in the medial or lateral compartment; however, a laxity difference by predominant knee compartment with OA was not statistically tested by any study in this systematic review. Frontal plane malalignment has been shown to be a driving factor in OA incidence and progression,²⁰ which is likely due to increased frontal

plane moments.^{5, 6, 59, 60} Consistently increased frontal plane moments may cause a progressive change in medial and lateral collateral ligament lengths, due to the increased internal forces necessary to balance the external moments.⁶¹ This change in collateral ligament lengths, depending on if the malalignment was varus or valgus, may affect the varus and valgus knee laxity differently but this relationship has yet to be established.

Females exhibited higher varus-valgus laxity compared with men in both OA subjects⁴¹ and controls.¹¹ While not part of this review, several studies indicate that females also have increased generalized laxity throughout all joints in the body compared with males,^{62–65} which may be related to hormonal status.⁶⁶ Hypermobility has been associated with OA^{67–69} and females have higher rates of hypermobility⁷⁰ and knee OA.⁷¹ Increased joint laxity may be an unmodifiable risk factor for women but more research is needed to rule out other sex differences as the cause of increased OA prevalence in females.

Function was quantified alongside varus-valgus laxity using clinical performance measures, chair-stand rate and 100 m walk test, and self-reported function, with the WO-MAC-PF and perception of instability. Clinical performance measures were tested in four studies,^{18, 37, 39, 48} but increased laxity was only significantly related to faster walking speed in one.³⁹ Self-reported activity limitations were also tested in these four studies, each resulting in a significant association with laxity,^{18, 37, 39, 48} however, the results were conflicting. Sharma et al found increased activity limitations in two studies^{18, 37} while van der Esch et al and Holla et al found reduced activity limitations with increased laxity.^{39, 48} These contrary results may be due to differences in subject populations, with one group based in the United States and the other in the Netherlands, but the influence of laxity testing device may also have an impact. van der Esch et al³⁹ designed their laxity testing device from Sharma et al,¹¹ but used a smaller magnitude of frontal plane knee moment during testing, 7.7 Nm versus 12 Nm. Even with a decrease in applied load, larger mean varus-valgus laxity was reported with this device compared with the two Sharma et al manuscripts. A device validation protocol would be necessary to identify the source of this variation. The only consistent finding was between varus-valgus laxity and self-reported instability, in which five studies found no significant relationship.^{35, 44, 45, 49, 53}

Limitations in current testing procedures have been identified during this systematic review and recommendations are listed to improve varus-valgus laxity measurement and reporting. The majority of varus-valgus laxity measurement techniques use a single applied load level to calculate varus and valgus laxity. The degree of laxity measured is inherently dependent on the load applied to the knee joint, however, the lack of a standard for applied load makes it difficult to compare or consolidate data across studies to perform any meta-analyses. A more detailed assessment of joint stability can be found by measuring applied force and relative tibiofemoral kinematics on continuous scales.^{56, 72} With this approach, load-displacement curves can be plotted and varus and valgus laxity can be found at any selected load level below that maximally applied. The increased radiation dose necessary to employ this technique when measuring joint space width using fluoroscopy is a major drawback, but for angular rotation measures of laxity it involves no increased risk to subjects. This method has the ability to characterize knee stability at multiple load points, and at specific varus or valgus knee angles to identify how joint stiffness changes with displacement. This technique would also

allow for comparisons and potential meta-analyses of laxity quantified at any load level below the maximum load applied.

There are inherent difficulties in accurately assessing varus-valgus laxity in vivo, namely applying the load to the femur and tibia and recording the joint kinematics under such load. Measuring varus-valgus knee angle can be difficult when the soft-tissue surrounding the joint is compressed during examination. Fluoroscopy can reduce the effect of soft-tissue artifact during measurement, but this approach generally only allows for a single camera viewpoint to make the knee angle determination (Telos Stress Device (METAX; Hungen, Germany)). It is also difficult to apply load directly in the varus-valgus direction, while completely avoiding any axial rotation of the femur or tibia. Internal-external rotation may appear to be varus-valgus rotation during laxity assessment and result in an inaccurate representation of anatomical varus-valgus laxity. Concurrently measuring femur and tibia kinematics with 6 degrees of freedom along with the applied load force vector during laxity testing⁷² would avoid this potential axial rotation problem. This approach allows for the calculation of applied varus-valgus load and motion in the anatomical reference frame. However, to accomplish this increase in precision motion capture, marker clusters are temporarily fixed to the femur and tibia. Rigidly fixing the markers to bone eliminates any effect of soft tissue artifacts and allows accurate measurement of internal-external rotation of the knee joint, but has been limited to intraoperative data collections during total knee arthroplasty due to its invasive nature.⁷²

Muscular guarding during varus-valgus laxity examination has been identified as one of the major sources of variation during a physical examination in subjects with OA.^{11, 32, 37, 56} Despite these concerns, attempts to control muscle activity were only mentioned in six of the included studies.^{11, 14, 31, 32, 37, 43} Two studies state that the testing device was designed to reduce incomplete muscle relaxation, but do not go into detail on how this was evaluated.^{11, 37} Three studies mention instructing the subject to relax completely during testing,^{14, 31, 32} but only one study attempted to measure the amount of guarding.⁴³ Lim et al identified irregular traces of varus-valgus angular displacement in real-time and repeated tests when they occurred. In addition, Lim et al suggests electro myography of muscles crossing the knee joint as a potential quality control measure during laxity testing, but this was not measured in any of the selected studies. It is possible that subjects with increased pain or higher perception of instability may guard their knees by cocontracting muscles more during testing, therefore making it difficult to gauge true passive laxity. Increased muscular cocontraction has been seen during dynamic motion in subjects with higher perceived instability⁴⁵ and similar cocontraction could be stabilizing the knee and reducing the magnitude of measured varus-valgus laxity.

Anesthetizing subjects before measurement is another potential solution that completely eliminates muscular guarding, although this adds significant complication to the measurement procedure. Subjects were under general anesthesia in three studies where varus-valgus laxity was measured in osteoarthritic knees prior to joint arthroplasty.^{36, 40, 42} Anterior knee laxity has been found to increase during unconscious measurement in subjects following anterior cruciate ligament disruption,⁷³ and it is plausible that varus-valgus laxity would increase as well when muscular forces crossing the knee are completely eliminated.

While recording, muscle activation would provide an estimate of muscular guarding, anesthetizing subjects may be the only way to eliminate muscular guarding during varus-valgus laxity testing. This technique is likely not reasonable as a standard of care for all patients with knee OA, however, may be necessary to eliminate the effect of muscle forces in research investigations.

All varus-valgus laxity research to date has measured laxity at a single time point in subjects with OA. Prospective studies are needed to identify how laxity changes with OA progression. This information is critical to identifying what compensations are necessary to function and how those affect disease development. This could potentially lead to alternative treatment strategies, such as surgical ligament balancing or bracing to manage patients with OA based on individualized laxity and biomechanical analysis.

Limitations of this review include those inherent in the reported studies, which were primarily observational in nature. There were inconsistencies in laxity definitions between studies and it was not always possible to identify when the same subjects were included in multiple manuscripts. Subject populations and measurement devices were also not homogeneous between studies. OA classification scales varied and the predominant knee compartment with OA was not consistently reported, which added to the difficulty merging subjects with similar disease states. The etiology of OA, whether traumatic or degenerative in nature, was not specified in the included studies and therefore could not be assessed. Lastly, there is potential for selection bias due to excluding studies that were not published in English, although subject populations from multiple continents are included in this review.

Conclusion

The results from the 40 articles included in this systematic review indicated that varus-valgus laxity was significantly larger in subjects with OA compared with controls in a majority of studies, and no study found laxity to be larger in controls. Varus-valgus laxity of the knee was investigated in subjects with OA and varying degrees of frontal plane alignment, disease severity, clinical performance, and self-reported function but no consensus finding could be identified. Females appear to have more varus-valgus laxity than males. Large variances in varus-valgus laxity were found between studies and this may be due to differences in testing device. Identifying standardized ways to measure load and varus-valgus displacement as continuous variables, along with controlling for muscular guarding, will help characterize all aspects of knee joint stability. This could potentially lead to meta-analyses and identify which aspects of knee joint stability are related to the incidence and progression of OA.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Fig. 1.

An illustration of a framework for the initiation and progression of osteoarthritis (I) Abnormal motion causes a shift in the contact location to a region not conditioned to high loads. Note: abnormal motion can result from a traumatic event such as anterior cruciate ligament injury or chronic changes to the musculoskeletal system that occur with aging (idiopathic knee). (II) Matrix damage to the superficial collagen network follows a shift to a load bearing regions that cannot adapt to changes in load bearing. (III) Friction increases following fibrillation of the collagen network. (IV) The maximum tangential force (F_t) that can be transmitted through the contact surface will increase following an increase in friction (IV). In addition, F_t will increase as the normal force increases. (V) Shear stress transferred

to the matrix will increase following degenerative changes and is dependent on the compressive force (F_n). (VI) Lowering the compressive force can lower the shear force transferred to the matrix and potentially slow the rate of cartilage degeneration. Reproduced with permission from Andriacchi et al.⁵

Fig. 2.

Flow diagram of selected studies for the systematic review. Notes: AP, anterior-posterior.

Table 1

Studies reporting angular laxity for OA and control groups. Results reported as mean (SD) unless noted otherwise

Author	Laxity measurement technique	OA groups	Control groups	OA quantified angular laxity (degrees)	Controls quantified angular laxity (degrees)
Brage et al 1994	Genucom Knee Analysis System 12.2 Nm load	Bilateral OA: <i>n</i> = 22 (43 knees) Mild = 10 Moderate = 15 Severe = 18	Age matched: <i>n</i> = 9 (18 knees)	Mild OA = 15 (4.8) Moderate OA = 10.9 (3.9) Severe OA = 10.4 (3.6)	Controls = 11.3 (3)
Wada et al 1996	Genucom Knee Analysis System 8 Nm load	Bilateral medial OA: <i>n</i> = 34 (68 knees) KL grade 1 = 15 KL grade 2 = 16 KL grade 3 = 19 KL grade 4 = 18	Elderly: <i>n</i> = 12 (24 knees)	KL grade 1 = 11.5 (5.4) KL grade 2 = 11.9 (4.3) KL grade 3 = 15.1 (5.1) KL grade 4 = 15.9 (5.4)	Controls = 12 (3.8)
Sharma et al 1999	Custom VV device 12 Nm load	Varied OA: <i>n</i> = 164 (328 knees) KL grade 0/1 = 39 KL grade 2 = 154 KL grade 3 = 84 KL grade 4 = 51	Older: <i>n</i> = 24 Young: <i>n</i> = 25	KL grade 0/1 = 4.9 (0.35) KL grade 2 = 4.4 (0.16) KL grade 3 = 5.1 (0.22) KL grade 4 = 5.7 (0.30)	Elderly controls = 3.4 (1.1) Young controls = 2.9 (1.0)
Wada et al 2002	Genucom Knee Analysis System 8 Nm load	Varied medial OA: <i>n</i> = 38 (38 knees) Prior to TKA	Age matched: <i>n</i> = 23	OA = 15 (7.9)	Controls = 12 (4.0)
Ishii et al 2009	TELOS VV stress radiograph 150 N load	Varied OA: <i>n</i> = 102 (120 knees) KL grade 2 = 1 KL grade 3 = 30 KL grade 4 = 89	Controls: <i>n</i> not reported	Median (25%, 75%) Varus laxity = 8 (6.9) Valgus laxity = 0 (0.2)	Median (25%, 75%) Varus laxity = 4 (3.4) Valgus laxity = 2 (0.25,3)
Creebly et al 2010	VV modified Kin-Com 12 Nm load	Varied Medial OA: <i>n</i> = 127 (127 knees)	Age matched: <i>n</i> = 32 (32 knees)	Mild OA: Varus = 10.5 (3.6)	Varus = 10.7 (3.7)

Author	Laxity measurement technique	OA groups	Control groups	OA quantified angular laxity (degrees)	Controls quantified angular laxity (degrees)
		Mild OA: <i>n</i> = 50 Moderate OA: <i>n</i> = 45 Severe OA: <i>n</i> = 32		Valgus = 9.6 (3.2) Total = 20.1 (6.4) Moderate OA: Varus = 9.2 (2.7) Valgus = 8.8 (2.4) Total = 18.0 (4.7) Severe OA: Varus = 8.5 (2.7) Valgus = 9.2 (2.9) Total = 17.7 (5.4)	Valgus = 8.4 (3.2) Total = 19.2 (6.5)
Sharma et al 2010	Custom VV device 12 Nm load	Varied OA: <i>n</i> = 950 (1,307 knees) KL grade 2 or 3 Alignment: Neutral: <i>n</i> = 232 Varus: <i>n</i> = 550 Valgus: <i>n</i> = 168	Without OA: <i>n</i> = 1,752 (2,958 knees) KL grade 0 or 1 Alignment: Neutral: <i>n</i> = 688 Varus: <i>n</i> = 725 Valgus: <i>n</i> = 339	Alignment: Neutral = 3.9 (2.5) Varus = 3.8 (2.5) Valgus = 4.4 (2.6)	Alignment: Neutral = 4.1 (2.6) Varus = 3.9 (2.5) Valgus = 3.8 (2.7)
Miyazaki et al 2012	Custom VV Stress Radiograph 22.1 Nm load	Bilateral medial OA: <i>n</i> = 46 (92 knees) KL grade 2 = 40 knees KL grade 3 = 32 knees KL grade 4 = 20 knees	Age matched: <i>n</i> = 22 KL grade 0 = 20 knees KL grade 1 = 24 knees	Preexercise KL grade 2 = 6.18 (1.78) KL grade 3/4 = 5.99 (2.81) Postexercise KL grade 2 = 8.85 (2.00) KL grade 3/4 = 8.55 (3.44)	Preexercise KL grade 0/1 = 6.98 (1.77) Postexercise KL grade 0/1 = 8.17 (2.18)
Chang et al 2014	Custom joint driving device VV ROM found when 8 and 12 Nm passive torque resistance reached	Varied medial OA: <i>n</i> = 14 (14 knees) KL grade 2 = 8 KL grade 3 = 6 Reported Instability: <i>n</i> = 9 (9 knees) KL grade 2 = 4 KL grade 3 = 5	Age matched: <i>n</i> = 14 (14 knees)	8 Nm OA total group = 4.13 (1.39) Unstable = 3.8 (1.2) Stable = 4.54 (1.75) 12 Nm OA total group = 6.83 (2.15) Unstable = 6.2 (2.0) Stable = 7.57 (2.44)	8 Nm 4.01 (1.52) 12 Nm 6.49 (1.92)

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Author	Laxity measurement technique	OA groups	Control groups	OA quantified angular laxity (degrees)	Controls quantified angular laxity (degrees)
		No instability: $n = 5$ (5 knees) KL grade 2 = 4 KL grade 3 = 1			

Abbreviations: KL, Kellgren–Lawrence; OA, osteoarthritis; ROM, range of motion; SD, standard deviation; VV, varus–valgus.

Table 2

Studies reporting joint space laxity for OA and control groups. Results reported as mean (SD) unless noted otherwise

Author	Laxity measurement technique	OA groups	Control groups	OA quantified joint space laxity (mm)	Controls quantified joint space laxity (mm)
Pai et al 1997	Method not reported Load not reported	Bilateral OA: <i>n</i> = 30 (60 knees) (8 M, 22 F) KL grade 2 = 20 KL grade 3 = 26 KL grade 4 = 14	Elderly: <i>n</i> = 29 (58 knees) (12 M, 17 F) KL grade 0 = 58	Med and lateral compartments combined. Bilateral OA: Right = 2.4 (1.8) Left = 2.2 (1.8)	Med and lateral Compartments combined. Elderly controls: Right = 1.1 (1.1) Left = 0.8 (1.2)
Sharma et al 1997	Method not reported Load not reported	Unilateral OA: <i>n</i> = 28 (28 knees) (13 M, 15 F) KL grade 0 = 6 unaffected KL grade 1 = 22 unaffected KL grade 2 = 7 KL grade 3 = 12 KL grade 4 = 9	Elderly: <i>n</i> = 29 (58 knees) (13 M, 16 F) KL grade 0 = 29	Med and lateral compartments combined. Unilateral OA: Affected limb = 3.1 Unaffected limb = 1.3	Med and lateral compartments combined. Elderly controls: Right = 1.3 Left = 0.8
Lewek et al 2004	TELOS VV stress radiograph 150 N load	Varied Med OA: <i>n</i> = 12 (6 M, 6 F)	Age matched: <i>n</i> = 12 (6 M, 6 F)	Med compart = 5.1 (1.5) Lat compart = 3.6 (1.6)	Med compart = 3.2 (1.0) Lat compart = 4.3 (1.3)
Lewek et al 2005	TELOS VV stress radiograph 150 N load	Varied Med OA: <i>n</i> = 21 (14 M, 7 F)	Age matched: <i>n</i> = 19 (12 M, 7 F)	Medial compart = 5.0 (1.7) Lat compart = 3.4 (1.7)	Medial compart = 3.3 (0.9) Lat compart = 4.1 (1.5)
Lewek et al 2006	TELOS VV stress radiograph 150 N load	Unilateral Med OA: <i>n</i> = 15 (30 knees) (9 M, 6 F)	Age matched: <i>n</i> = 15 (30 knees) (9 M, 6 F)	Medial compart = 4.9 (1.8) Lat compart = 3.5 (1.5)	Med compart = 3.2 (0.9) Lat compart = 4.0 (1.4)
Rudolph et al 2007	TELOS VV stress radiograph 150 N load	Varied Med OA: <i>n</i> = 15 (15 knees) (8 M, 7 F)	Middle aged: <i>n</i> = 15 (15 knees) (8 M, 7 F) Older: <i>n</i> = 14 (14 knees)	Medial compart = 4.77 (1.72) Lat compart = 3.56 (1.65)	Middle age controls: Med compart = 3.12 (0.95) Lat compart = 4.20 (1.27)

Author	Laxity measurement technique	OA groups	Control groups	OA quantified joint space laxity (mm)	Controls quantified joint space laxity (mm)
Schmitt et al 2007	TELOS VV stress radiograph 150 N load	Varied Medial OA: <i>n</i> = 28 (28 knees) (14 M, 14 F) KL grade 2 = 17 KL grade 3 = 8 KL grade 4 = 3	Age matched: <i>n</i> = 26 (26 knees) (13 M, 13 F) KL grade 0 = 26	Mean (95% CI) Medial compartment = 4.23 (3.57, 4.89) Lat compartment = 2.77 (2.28, 3.27)	Older controls: Med compartment = 3.05 (0.76) Lat compartment = 3.63 (1.34) Mean (95% CI) Med compartment = 2.76 (2.38, 3.14) Lat compartment = 3.52 (3.04, 4.00)
Kumar et al 2013	TELOS VV stress radiograph 150 N load	Varied Medial OA: <i>n</i> = 16 (16 knees) (8 M, 8 F) KL 2	Controls: <i>n</i> = 12 (12 knees) (6 M, 6 F) KL 1	Mean (95% CI) Med laxity = 5.5 (4.6, 6.4) Lat laxity = 3.4 (2.7, 4.1)	Mean (95% CI) Med laxity = 3.3 (2.4, 4.2) Lat laxity = 4.7 (3.2, 5.1)

Abbreviations: CI, confidence interval; Compartment, compartment; F, female; KL, Kellgren–Lawrence; Lat, lateral; M, male; Med, medial; OA, osteoarthritis; SD, standard deviation; VV, varus-valgus.

Note: Results are reported as mean (SD) unless noted otherwise.

Table 3

Overview of findings

Topic	Total no. of studies	Study findings	Qualitative summary
OA vs controls	17	11 studies found increased laxity in OA vs controls	11/15 studies found a significant difference between groups when tested
OA severity	7	3 studies showed differences between OA severities with conflicting results	No consensus could be reached
Alignment	4	2 studies found altered laxity depending on alignment, but no common finding	No consensus could be reached
Clinical and self-reported function	18	4 studies found conflicting results between laxity and clinical performance and self-reported activity limitations. 5 studies found laxity was unrelated to perceived instability.	No consensus could be reached with varus-valgus laxity and clinical performance or activity limitations. Varus-valgus laxity was unrelated to perceived instability in 5 studies.
Sex differences	1	Females found to have more varus-valgus laxity than males	Only 1 study tested varus-valgus laxity in OA males vs OA females, but females have been found to have higher laxity in other populations and joints