# Kinematics of the Spine Under Healthy and Degenerative Conditions: A Systematic Review

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**Abstract**—Understanding spinal kinematics is essential, not only for the comprehension and diagnosis of spinal diseases, but also for improving modern tools and software. The sheer volume and complexity of now available information can be overwhelming. We aimed to distil it into a form that facilitates comparison among diverse studies addressing spinal kinematics under healthy and degenerative conditions. We specifically aimed to define a baseline definition of the spectrum of normal spinal kinematics that in turn allows a comparable definition of kinematics of the degenerative lumbar spine. The considered data was obtained by a systematic MEDLINE search including studies on angular/translational segmental motion contribution, range of motion, coupling and center of rotation. As for degenerative conditions, we collected publications on disc degeneration, facet joint osteoarthritis, facet joint tropism, spondylolisthesis, ligament degeneration and paraspinal muscle degeneration. While we could demonstrate repeating motion patterns for some topics, agreement in other fields is limited due to methodological variances and small sample sizes, particularly in publications with highly accurate but complex techniques. Besides, the high frequency of concurrent degenerative processes complicates the association between diseases and subsequent kinematical changes. Despite several substantial gaps, we stand at the precipice of technological break-throughs that can power future large-scale studies.

**Keywords**—Motion, Flexion, Extension, Axial rotation, Lateral bending, Center of rotation, Range of motion.

## ABBREVIATIONS

| | |
|---|---|
| AOR | Axis of rotation |
| AR | Axial rotation |
| COR | Center of rotation |
| DD | Disc degeneration |
| FE | Flexion/extension |
| FJOA | Facet joint osteoarthritis |
| FJT | Facet joint tropism |
| FU | Functional unit |
| IL | Interspinous ligament |
| LB | Lateral bending |
| LD | Ligament degeneration |
| LF | Ligamentum flavum |
| LFH | Ligamentum flavum hypertrophy |
| NZ | Neutral zone |
| OA | Osteoarthritis |
| PMD | Paraspinal muscle degeneration |
| ROM | Range of motion |
| SL | Spondylolisthesis |

## INTRODUCTION

Kinematics of the lumbar spine is a matter of great interest in the field of spinal orthopedics and reason for countless investigations during the last decades. A profound understanding is essential for not only the comprehension and diagnosis of numerous spine diseases, but also for the improvement of tools, such as motion sustaining products and prostheses. Modern implants usually aim at restoring healthy kinematics, which of course requires precise knowledge of these complex relations. The same is true for multibody

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dynamics software using kinematical data as input for simulations.

Discussing the kinematics of the "healthy" spine inevitably raises the question of its definition and whether it is possible to clearly distinct a healthy spine from a pathologically behaving exemplar by just studying its motion pattern. This could serve as a tool for diagnostics and disease classification. Several approaches in this direction have been taken, for example in order to diagnose spondylolisthesis (SL) by evaluating segmental instability[62,104] or to assess the degree of disc degeneration (DD) by measuring the shift of the center of rotation (COR).[37,38,103,119] Multiple authors tried to come up with a definition of spinal instability. "Damage at one of the three spinal columns",[40,42] hyper-translational and hyper-angular motion[14,48,52,83,126–128] or even a disordered COR[22,104,119] were held responsible for a spinal imbalance. However, a useful classification system could not yet be established in the clinical practice. Besides, the applied methods for studying healthy as well as pathological motion patterns of the spine are extremely diverse and range from simple static end-position image comparison to sophisticated techniques like 3D motion reconstructions. Moreover, test procedures as well as experiment parameters are manifold. This adds up to an immense diversity of outcomes within literature, often even contradicting. Some of the widespread, fundamental principles in the field are therefore based on single publications or even originate from *in vitro* tests, and even though more recent publications indicate contradictory results, these principles are not discarded.

Driven by the lack of clarity in this field, we attempt to clear the fog as far as possible by providing an extensive overview on published literature in the field of spinal kinematics under healthy and degenerative conditions. We further try to find explanations for contradictory results and point out gaps in literature, in order to inspire and motivate research in these fields. In addition, this review shall serve as a reference and foundation for comparisons with any kind of existing or future results. Literature has grown substantially since 1977, when White and Panjabi published a first review on the basics of spinal kinematics.[140] Based on their work,[140,141] we analysed the literature on the following topics: angular segmental contribution, translational segmental contribution, maximal range of motion (ROM), coupling, COR and phase lag (PL). In addition, we summarized publications on spinal kinematics in DD, facet joint osteoarthritis (FJOA), facet joint tropism (FJT), SL, ligament degeneration (LD) and paraspinal muscle degeneration (PMD). Since most pathologies show the highest prevalence in the lumber spine, we focused on this lower section of the spine.

## MATERIALS AND METHODS

MEDLINE was searched with predefined keywords for each topic individually, leading to 2992 angular/translational segmental motion contribution (Fig. 12), 2992 coupling (Fig. 14), 2992 maximal ROM (Fig. 13), 1054 COR (Fig. 15) and 2992 PL (Fig. 16) hits. Similarly, 763 DD: mechanical stiffness (Fig. 17), 2688 DD: ROM (Fig. 18), 397 FJOA (Fig. 19), 38 FJT (Fig. 20), 283 SL (Fig. 21), 119 LD (Fig. 22) and 157 PMD (Fig. 23) publications were found for our section on pathologies. These results were then narrowed with inclusion and exclusion criteria and supplemented with a reference search. All papers from 1970 to 2016 were included. Cadaver studies were excluded, since they neglect neuromuscular interaction, which is one of the most essential components defining spinal *in vivo* behavior. However, as indicated there, they were included in the section on COR since no *in vivo* studies were found for kinematical behavior during lateral bending (LB) and axial rotation (AR).

Eventually, this resulted to a final selection of 36 angular/translational segmental motion contribution (Fig. 12; Table 1), 9 coupling (Fig. 14; Table 3), 11 maximal ROM (Fig. 13; Table 2), 15 COR (Fig. 15; Table 4) and 9 PL (Fig. 16; Table 5) publications. 9 DD: mechanical stiffness (Fig. 17; Table 6), 13 DD: ROM (Fig. 18; Tables 7; 8), 4 FJOA (Fig. 19; Table 9), 1 FJT (Fig. 20; Table 10), 17 SL (Fig. 21; Table 11), 3 LD (Fig. 22; Table 12) and 1 PMD (Fig. 23; Table 13) studies were selected for the part on pathologies. A detailed description of the applied search strategy and precise literature overview is provided in the Appendix.

Results of the final literature selection were brought into a comparable form. All results dealing with segmental contribution were converted into percentages, relative to the overall motion from L1 to S1. When data was missing for certain segmental levels, an average value from all available percental values on that level was taken and visualized with a dashed line. Results that were separated into groups according to age, sex or other factors were averaged. For left and right LB and AR pooled values were used. Additionally, statistical error propagation was considered when pooling standard deviation values.[26]

Values and plots in the sections on pathologies are normalized to values measured under healthy conditions, in order to allow comparisons.

## HEALTHY CONDITIONS

The lumbar spine is an essential contributor for bending and twisting movements during daily activities. Besides, lumbar joints are also exposed to high loads, leading to increased susceptibility for many degenerative diseases, such as DD or FJOA.[114] On these grounds, the most caudal section of the spine is object of numerous investigations and in focus of the spinal research.

As previously mentioned, methodical strategies are various (methodologies are listed in the Appendix), many parameters have to be defined. While load bearing[5] does not seem to influence the kinematic and movement direction (forward vs. backward flexion) has an only minor effect,[3] movement execution (passive vs. active)[24] significantly alters the outcome. Besides, experimental factors like movement velocities (arbitrary vs. prescribed) and ROM (restricted vs. the maximum possible) and body posture need to be investigated as potential influencing factors.

Aside from experimental parameters, patient populations are considerably different amongst the authors. Age seems to have an only minor effect and only age groups above 50 years seem to have an altered kinematics.[48,143] Gender[23,143] and body weight[48] was shown to be inconsequential for motion patterns.

### Segmental Motion Contribution

#### Angular

The lumbar spine covers substantial ROMs during normal daily activities. The overall movement, which will be referred to as 100%, is the sum of smaller movements of all the segments from L1 to S1. The contribution of these segments to the overall movement however varies considerably and depends on the nature of the performed movement.

Angular motion contribution for flexion/extension (FE), LB and AR is discussed in the following, defined as the angular, relative contribution of one segment to the overall movement of the lumbar spine (L1 to S1).

It is derived from calculating the difference of the intervertebral angle between two or more positional frames. In 2D studies (planar X-ray, video fluoroscopy, *etc.*), the intervertebral angle is measured between the lower endplate of the supraadjacent vertebrae and the upper endplate of the subjacent vertebrae. In 3D, the relative rotation of two vertebras is determined by calculating the rotational matrix that brings a vertebra from one into the other position.
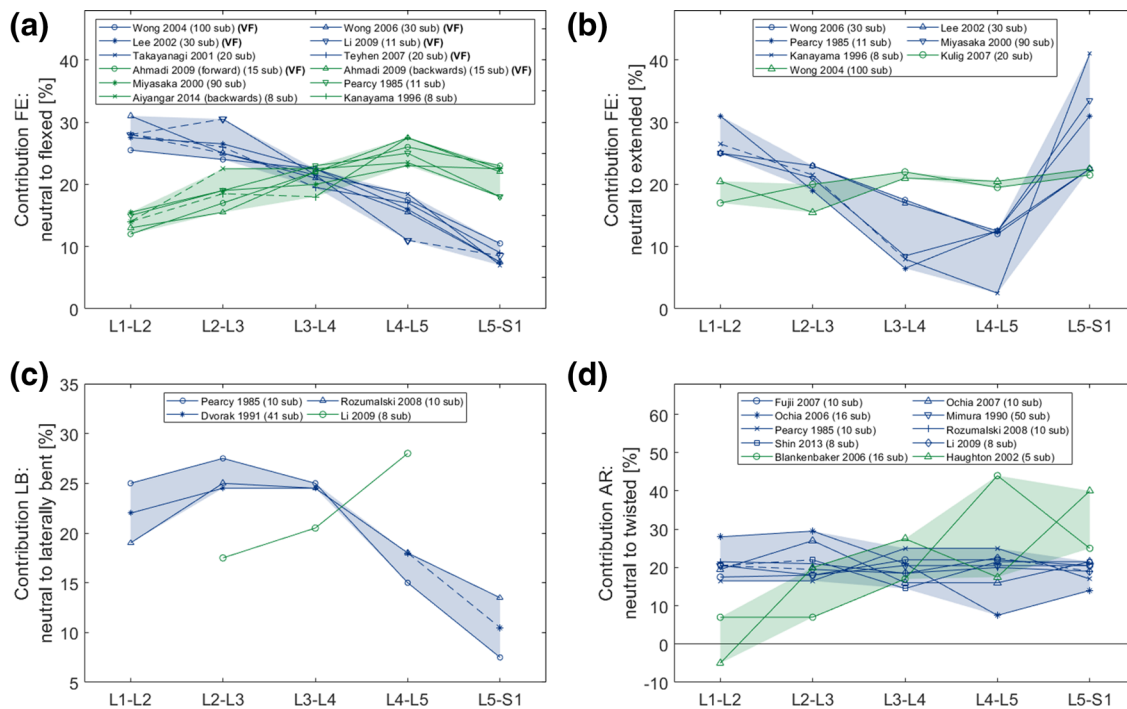


**FIGURE 1.  Angular contribution of the segments. (a) Flexion, (b) extension, (c) lateral bending, and (d) axial rotation. Dashed lines are used when the values for certain levels were not available and an average value was used. Shaded areas indicate a classification according to patterns that are further discussed in the main text. Negative values in (d) represent counter-rotating movement. Complementary to the content of the main text, the studies which used pure biplanar video-fluoroscopic are indicated with (VF) in the legend of plot (a).**

*Flexion/Extension* Analyzing spinal movements in the sagittal plane (neutral to flexed) has yielded two major contribution patterns in literature. A cranio-caudally decreasing[70,71,127,132,142,143] and a cranio-caudally increasing contribution pattern[3,7,56,83,100] with a slight drop at the L5–S1 segment (Fig. 1a).

These two patterns seem to be completely conflicting at first sight. However, according to our understanding, there are at least two possible explanations:

- *Angular Contribution is ROM Dependent* All studies printed in blue (decreasing pattern) performed their experiments with a limited ROM of either 35,[132] 40[70,142,143] or 45 degrees of flexion.[71] Takayanagi et al.[127] did not limit the ROM, they however performed their experiments in a sitting position, which automatically limits the movement. In contrast, all experiments printed in green (increasing pattern) were performed with an unlimited magnitude of flexion.

This finding implies that the angular contribution could be ROM dependent, which is in line with Miyasaka et al.[83] They investigated ROM dependent segmental contribution in 90 subjects and documented a higher contribution of the cranial segments at the initial stage of flexion and the typical L4–L5 peak pattern at the maximal magnitude.

However, Aiyangar[7] and Ahmadi[3] (which both found an increasing pattern with a drop at L5–S1) show their data over the complete ROM from neutral position to the maximal flexion, and in addition some stages in between. In both studies, no difference in contribution patterns between the intermediate stages and the end of the movement could be observed.

- *Posture* Most studies printed in blue (decreasing pattern) used pure video-fluoroscopic (VF) experiments,[70,71,132,142,143] either uni- or biplanar. In contrast, most other studies (increasing pattern) used different experimental techniques (except Ahmadi[3]). In order to place hands and arms outside of the continuously recorded frame, subjects are usually asked to cross their hands behind their head during videofluoroscopy, which will likely influence the motion pattern. Crawford and Jull[19] showed a 50% reduced thoracic extension during bilateral arm elevation. A similar effect can be expected for the lumbar spine. An additional explanation is that the initial (neutral) position is different when arms are elevated (hollow back).

We again detected two patterns for sagittal extension (neutral to extended position): one suggesting roughly uniform contribution (green)[66,143] and the other with high contribution of the cranial and caudal segments and a drop at the intermediated levels (blue)[56,70,83,100,142] (Fig. 1b). Importantly, Kulig et al.[66] (uniform pattern) put their subjects in a prone position and extension was achieved by actively pushing the trunk off the ground. This certainly im-

pacts spinal kinematics. After exclusion of this study, the majority of data supports the parabolic pattern during sagittal extension. A direct ROM dependency as it is assumed for inclination does not seem to exist for extension, a finding that is in line with Miyasaka.[83]

*Lateral Bending* Contribution patterns for LB are relatively consistent among the authors,[23,100,112] with the exception of Li et al.[71] (Fig. 1c). Otherwise, the findings indicate a contribution increase from L1 to L3, with a peak at the L2–L3 level and a monotonical decrease from L3 to S1 (Fig. 5b). Interestingly, Simon[125] showed a similar distribution for the gap between the cartilage endplates of the facet joints. We accordingly presume that LB is primarily limited by the ROM of the facet joints.

*Axial Rotation* For AR, again, two patterns occur depending on the ROM (Fig. 1d). Blankenbaker[13] and Haughton[46] let their subjects rotate from a neutral position to a limited ROM of only 8° (green, increasing pattern). Studies showing a uniform pattern (blue) measured at either 45°[31] or 50°,[86,87] or even at the maximally possible torsion.[71,80,100,112,123] Taken together, this implies that the lower segments mainly perform the initial phase of AR whereas participation of each level is very similar for larger torsions.

Interestingly, facet joint orientation is also parabolically distributing[99]: the joints of the inferior and superior lumbar segments are more coronally aligned than the joints of the intermediate ones. Coronal alignment, as opposed to more sagittal alignment, is less restricting for AR. Distributions of joint orientation and gap width therefore cause opposing impacts on the ROM: alignment of the joints favors movement in the inferior and superior segments, whereas the increased gap widths of the intermediate segments, as described above, facilitate motion of the levels in between. This could explain the approximately flat curve of the segment contribution during AR.

### Translational

Translational contribution is defined as the percental translational movement of a vertebra, in relation to the sum of all lumbar translations. It is measured in anterior–posterior direction for FE and AR, in left–right direction for LB.

In 2D experiments, segmental translation is measured as the difference in horizontal (meaning in direction of the superior endplate of the subadjacent vertebra) distance between the posteroinferior corner of the supraadjacent vertebra and the posterosuperior corner of the subjacent vertebra. In 3D experiments, translation is defined as the motion of the cranial vertebral coordinate system origin (center of vertebra) in the caudal vertebral coordinate system. Therefore,

2D and 3D values are not exactly identical; they will however yield similar results.

*Flexion/Extension* Data for translational contribution can be grouped in two motion patterns very similar to the patterns for angular contribution during flexion (Fig. 2a). With the exception of Li *et al.*,[71] the studies either show a monotonically descending curve (blue)[127,132] or a slightly ascending graph with a drop at L5–S1 (green).[3,7,56] Only one study could be found for extension (Fig. 2b).[56]

Interestingly, this correlation between angular and transitional contribution in the sagittal plane indicates that intervertebral discs are simultaneously exposed to high angular deflections as well as anteroposterior loads. Such double loads significantly intensify the stress at highly contributing levels (e.g. L4–L5) and might be the reason for the higher prevalence of degenerative changes at these segments.[114]

*Lateral Bending/Axial Rotation* Literature for translational contribution in LB is scarce.[23,71] However, some similarity to the pattern for angular contribution (Fig. 1c) can be seen (Fig. 2c). This is not true for AR, showing a cranio-caudally decreasing contribution pattern for translation (Fig. 2d).[71,86,87]

### Maximal Range of Motion

Hayes *et al.* first published values for lumbar ROM in 1989, and their results are still used as reference values today.[48] However, much research has been done in the meantime. For this review, we only considered studies performing end-to-end movements (meaning the maximal possible movement from flexion to extension, from left to right bending and from left to right twisting) in order to retrieve maximal values for the segmental ROM. Results are shown in Fig. 3, illustrating only moderate agreement between different authors.[14,23,29,48,71,80,100,112,123,128]

Literature for maximal translational movement is scarce. Again, measuring methods are considerably different and do not allow comparisons between different publications.

### Coupled Motion

As already observed in 1966,[110] spinal motion around one axis is always accompanied by coupled translational and rotational components around or along other axes. This phenomenon is caused by our spine, whose curvy shape prohibits pure uniaxial motion.

Literature on coupling motion reports very conflicting results, mainly due to differing definitions. For example, it is essential whether rotation is measured around an inertial or a local coordinate system, since coupled motion is rotation that appears during the spinal rotation in primary direction. Besides, since the results usually arise from newer methods with spatial
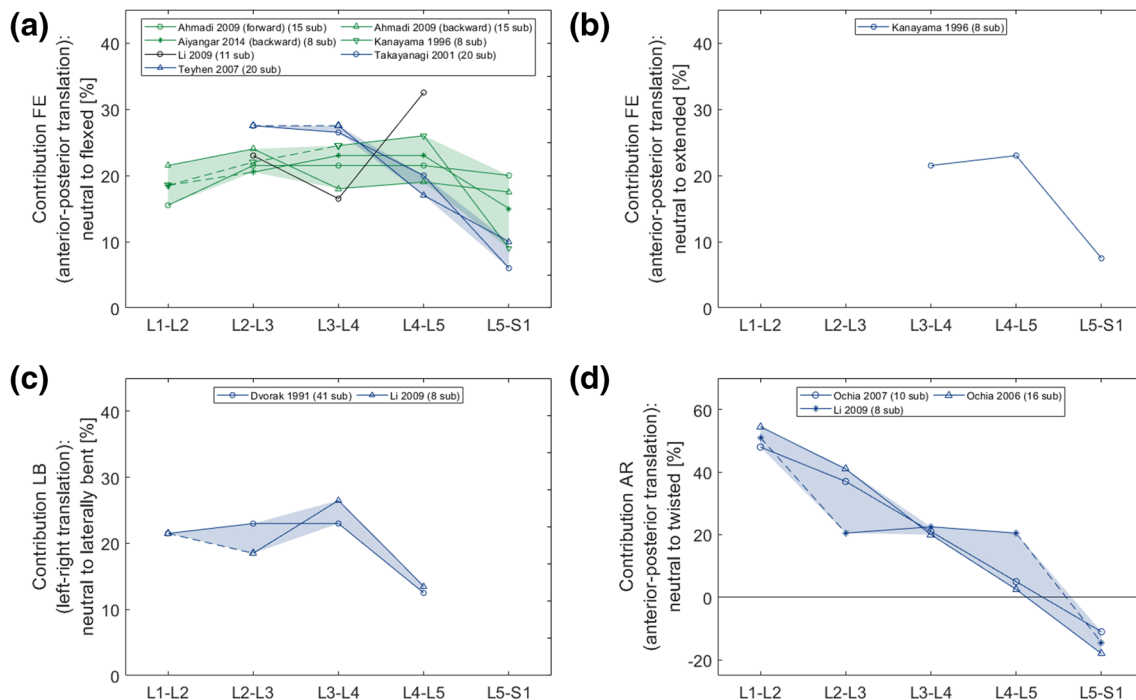


FIGURE 2. Translational contribution of the segments. (a) Flexion, (b) extension, (c) lateral bending, and (d) axial rotation. Dashed lines are used when the values for certain levels were not available and an average value was used. Shaded areas indicate a classification according to patterns that are further discussed in the main text.
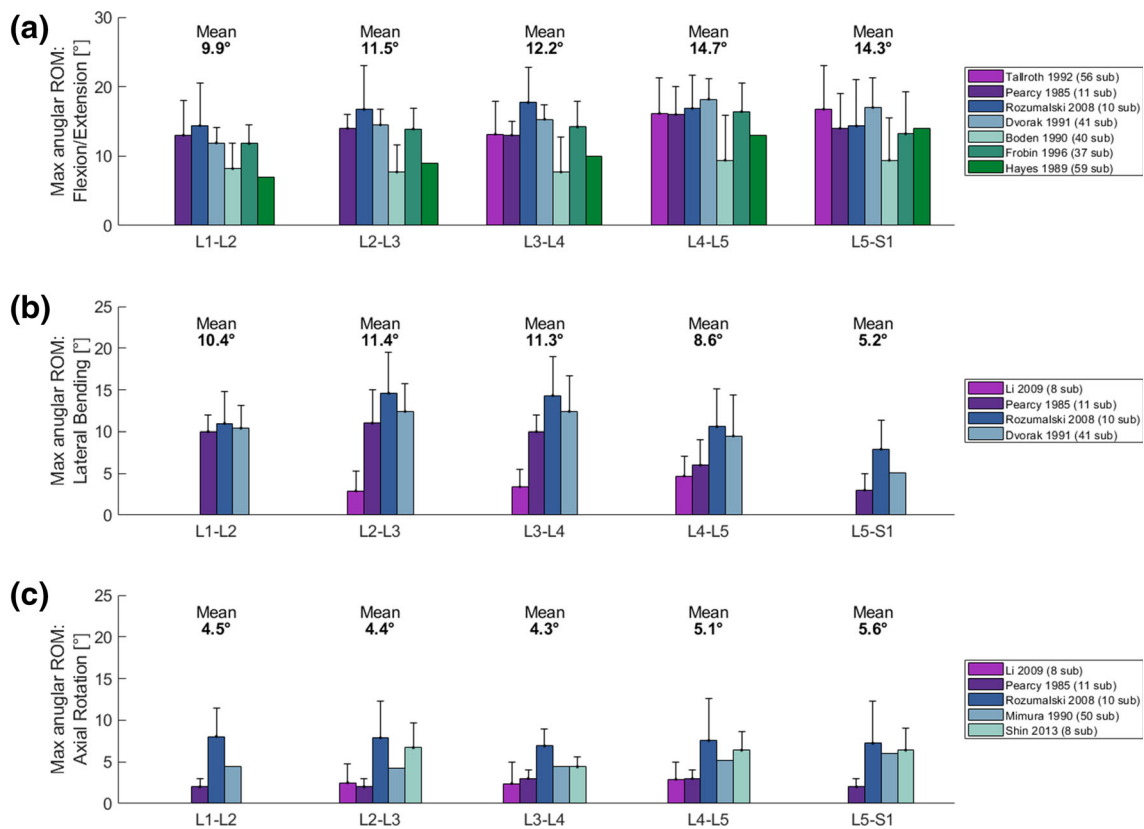
FIGURE 3. Maximal angular ROM for each lumbar segment. (a) Flexion/extension, (b) lateral bending, and (c) axial rotation. Means are weighted averages (in relation to the number of subjects). Error bars represent the standard deviation (error information was not available for all the shown data from Hayes *et al.*[48] and Mimura *et al.*,[80] as well as for the ROM measured by Dvorak *et al.*[23] during lateral bending in the segment L5–S1).

vertebrae tracking, they strongly depend on the convention of rotation order (e.g. Euler formulation, Roll-Nick-Gier, *etc.*). This information is usually lacking and comparisons are difficult.

Nevertheless, Fig. 4 gives an overview of the published data in literature.[31,71,86,87,112,123,145] Interestingly, coupled motion during FE (Fig. 4a) ranges up to 60% (considering the very large values from Li[71] as outliers), even though the centers of mass in the coronal plane are approximately aligned above each other and one would therefore not expect to find any substantial motion around other axes.

Coupled motion during LB was measured to be between 40 and 100% of the primary motion (Fig. 4b) and ranged from 100% up to 400% for AR (Fig. 4c). This is comprehensible, given the fact that the centers of mass are not aligned in the sagittal and axial plane.

### Center of Rotation

The COR is generally defined as the penetration point of the axis of rotation (AOR) in the plane of motion. Whilst trying to characterize pathological kinematics, several authors suggested the COR to be an important indicator for early progression of spinal diseases.[21,38,69,88,111,119] However, its description is challenging and associated with some inaccuracies, such as a large error magnification,[93] projection errors due to coupled motion and calculation method dependence in 3D.[136]

### Flexion/Extension

Figure 5 displays the measured CORs from four publications[6,88,101,148] in either extension (black), neutral position (grey) or flexion (white). The mean COR of a face joint is usually located in the posterior, upper quarter of the caudal vertebra or in the posterior, lower quarter of the intervertebral disc. It almost completely stays in the intervertebral space for L5–S1.

The COR tends to travel from dorsal to ventral and even slightly from caudal to cranial during inclination. Additionally, two groups showed a loop-shaped COR path at certain levels,[6,88] which is in line with an *in vitro* study by Seligman *et al.*[119]
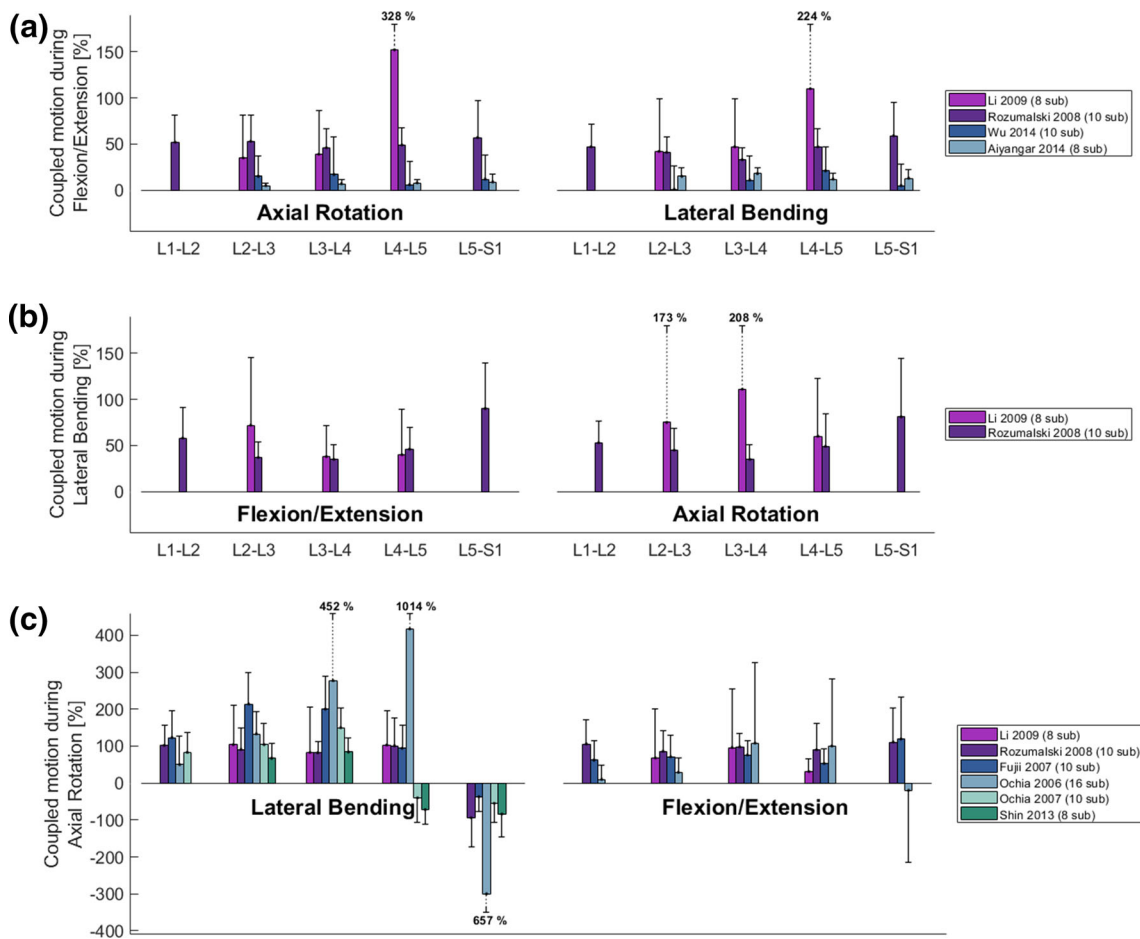
FIGURE 4. Coupled motion [as ratio (%) of the maximal ROM of the primary motion]. Negative values appear due to motion to the opposite direction. Values exceeding 100% are larger than movement in the primary direction. (a) Coupled motion during flexion and extension, (b) coupled motion during lateral bending, and (c) coupled motion during axial rotation. Error bars represent the standard deviation.

Importantly, all the illustrated CORs are mean values. Pearcy,[101] Yoshioka[148] and Aiyangar[6] illustrated COR spreading on areas sometimes larger than the vertebral body projection.

### Lateral Bending

No *in vivo* study for LB could be found. *In vitro* studies suggest the COR to be located in the ipsilateral posterior disc region.[107,111,141]

### Axial Rotation

One *in vivo* study for AR could be detected.[146] They calculated the COR for maximal magnitude twisting and found it to be located ventrally of the vertebral body, close to the center of the trunk. As opposed to this, *in vitro* studies usually indicate the COR in the posterior part of the vertebra, posteriorly shifting with increasing torque.[107,117,124,137] Xia assumed that this is because the facet joints are not the motion-limiting factor under *in vivo* conditions (as it is the case *in vitro*), but rather the surrounding tissue.[146]

### Phase Lag

The term "phase lag" describes a shift of motion among the vertebras. PL occurs when segments start the initial movement with a certain time delay or arrive at the final position with a certain time offset. Whether this phenomenon exists or not and to what extent it exists is controversy discussed in the literature. Kanayama[56] and Harada[43] experienced a shifted onset from cranial to caudal. Okawa[89] experienced both, a sequential and a simultaneous motion pattern for pathological subjects. Ahmadi[3] and Takayanagi[127] found onset delay at the caudal L5–S1 level for some subjects. Aiyangar's[7,8] studies imply a varying contribution of the segments at the beginning or end of motion. All other authors do not see (Lee,[70] Wong,[142] Wong[143]) or mention (Teyhen[132]) a sequential pattern.
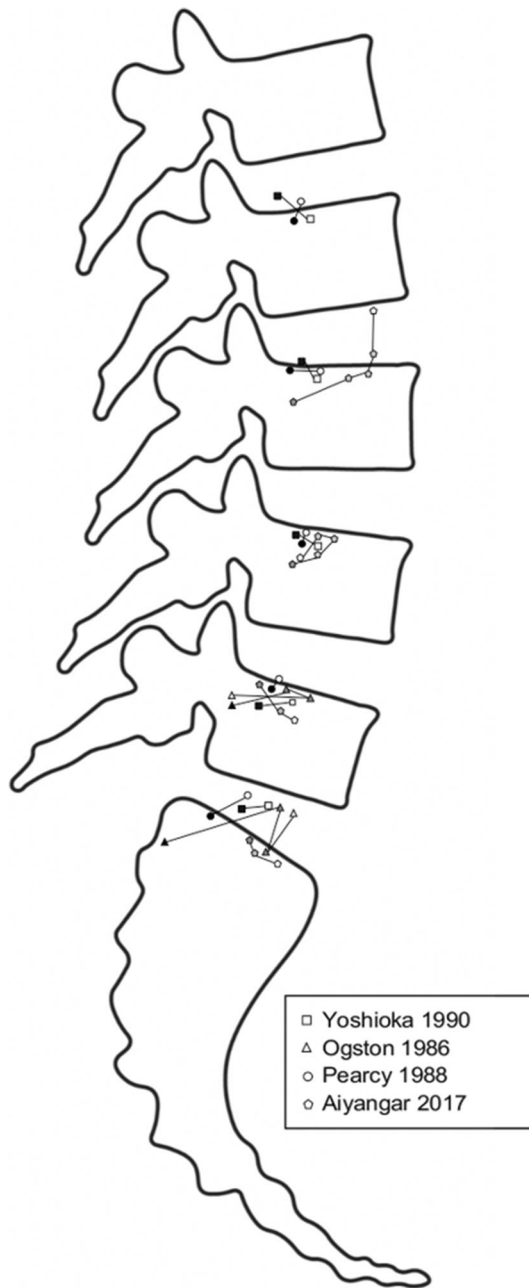
FIGURE 5. Center of rotation for *in vivo* data from extension (dark fill), to neutral (grey fill), to flexion (white fill) (data illustration was done by roughly redrawing the figures provided in the corresponding papers[6,88,101,148]).

An interesting observation is that most studies which experiences PL were performed with hip restriction or hip support. On the other side, in most of the other studies the subjects were free to move. However, this is not true for every case.[127,132] Never-

theless, pelvic restriction might be a potential explanation for the occurrence of sequential onset as it could change the initial neutral position.

### Flexion/Extension

Kanayama,[56] Harada[43] and Okawa[89] did investigate the sequential onset for forward and backward flexion. They experience that segmental motion during forward flexion starts from the upper cranial levels and sequentially progresses downwards. For backward flexion, they observe the opposite, PLs starting from the most caudal L5/S1 level.

## DEGENERATIVE CONDITIONS

Degenerative diseases of the lumbar spine are difficult to diagnose, and even more challenging is their classification. Clear associations between diseases or disease stages and certain motion patterns would facilitate both diagnostics and treatment. However, in many cases, spinal pathologies are not isolated phenomena, but are accompanied by other degenerative changes.[53,115,120] However, most of the studies classify patients according to the state of their primary disease, neglecting accompanying changes that are also likely to influence the kinematical behavior. Besides, even though structural changes follow a certain progress pattern,[32] it is challenging to ambiguously assign them to a certain state of disease. Various grading systems are applied, and depending on the imaging technique, patients are assigned to different progress stages. Comparability is therefore limited.[11,12,28,141,149]

### Disc Degeneration

Despite extensive research, the etiology of lumbar DD is not fully understood. In an extensive review, Hadjipavlou *et al.*[39] conclude that the disease is initiated by a complex, multifactorial combination of stimuli such as ageing, nutrition supply and mechanical factors (vibration, torsion, compression, shear). Details however remain unclear, and kinematics are therefore even more difficult to assess.

### Mechanical Stiffness

Even though not directly related, it is conceivable that the mechanical stiffness of the discs plays a role in spinal kinematics. Usually, it is described with a load–deformation curve,[94] plotting the moment to the dis-

placement at each time point when bending a functional unit (FU). Load–deformation curves consist of a neutral zone (NZ), where deformation occurs against very little internal resistance, and an elastic zone, where approximately linear resistance occurs (stiffness). Besides, the ROM is shown as the total deformation upon maximal loading. The form of the NZ describes a materials laxity, whereas the ROM is an indicator for the overall stiffness.

We summarized the findings of different *in vitro* studies in the following, detailed results are listed in the Appendix. However, it has to be taken into account that different grading systems and loading conditions were applied.

*Flexion/Extension* Two different patterns could be detected. Some studies found decreasing ROMs for advanced stages of DD, indicating stiffening,[60,81,85,150] others showed the often-cited pattern described by Kirkaldy-Willis[61]: an initially increasing ROM due to instability, followed by a loss of flexibility in the final stage.[32,81,141] Tanaka et al.[130] found both patterns and suggested level dependency (the decreasing pattern for the cranial levels and the increasing–decreasing one for the caudal levels).

However, most studies agree on a broadening NZ for progressed stages of DD, indicating increased laxity.

*Lateral Bending* Associations between FE and LB behavior are frequently seen, implying that DD has similar impacts on both. However, most authors report an initial broadening and subsequent narrowing of the NZ, suggesting a decrease in laxity.

*Axial Rotation* Most authors agree on a strong increase for both ROM and laxity during progress of DD.

Taken all together, the data is contradictory and rather implies an arbitrary mechanical behavior than a predetermined pattern. This is not particularly surprising, given that DD is probably a complex set of differently directed processes. Dehydration of the nucleus pulposus, osteophyte formation and diffuse sclerosis are associated with increased stiffness and hence a decrease in flexibility. On the other hand, tendon laxity due to disc height loss and facet joint arthrosis, leading to a worsened ability to guide movement, cause increased segmental mobility.

Beside the complex etiology, Tanaka et al.[130] suggested level dependent kinematical behavior, which is otherwise insufficiently investigated to date. However, differing stiffness values between the levels were already shown in 1994.[97]

We conclude, that it is not exclusively the mechanical properties of a disc that influences the spinal kinematics, but that disc mechanics may alter motion patterns and is therefore worth considering in this context. We could not find any correlation between the measurement results of the different authors and their applied loading conditions, grading system or any other experimental factor. This finding is in line with a previously published review.[36]

### Angular Range of Motion

DD changes the kinematics of the affected levels and it is therefore reasonable to expect differing ROMs. *Flexion/Extension* Figure 6 illustrates the change in segmental ROM in the sagittal plane, depending on the severity of DD. Agreement amongst the authors[58,64,67,133] for the individual levels is lacking, however, they almost perfectly coincide when it comes to the overall change in segmental ROM (Fig. 6a). This might be explained by the larger data set of the combined data, which is less sensitive to the spread.

Figure 6a demonstrates that segmental mobility stays relatively constant until the final stage, where mobility drops down to below 50% of the healthy values. Thus the question arises whether and where the lost motion gets compensated, reminiscent of the still on-going debate about how spinal fusions influence spinal kinematics.[73] According to Lee et al.,[68] although all of the segments somehow participate, the significant part of motion is compensated in the L1–L2 segment (in patients with DD in L4–L5 or L5–S1).

*Lateral Bending/Axial Rotation* No literature could be found for LB. For AR, Basques et al.[10] was the only group investigating DD-associated ROM change *in vivo*. According to them, segments slowly lose their ROM during the progress of DD, with the strongest decrease between stages 4 and 5.

*Overall ROM* Most studies could not demonstrate any significant change in overall lumbar ROM.[30,55,76,108,134] However, Weiner et al.[139] found decreased ROM in forward and lateral flexion, and Lee et al.[68] demonstrated decreased lumbar ROM for patients with stage 4 or 5 DD and $< 5°$ motion in L4–L5 or L5–S1. This implies that even though lost motion is partly compensated by the other segments (as described above), overall lumbar ROM is also decreased. The reason why many authors could not find significant motion reduction in overall lumbar motion is probably because they classified the segments according to existence or non-existence of DD and did not consider whether the degenerated segments indeed had a reduced mobility.

### Translational Range of Motion

Similar as for the angular ROM, consensus between the authors could only be found for the mean segmental change in translational ROM (Fig. 7).[9,58,64] After an initial increase at the intermediate stages,
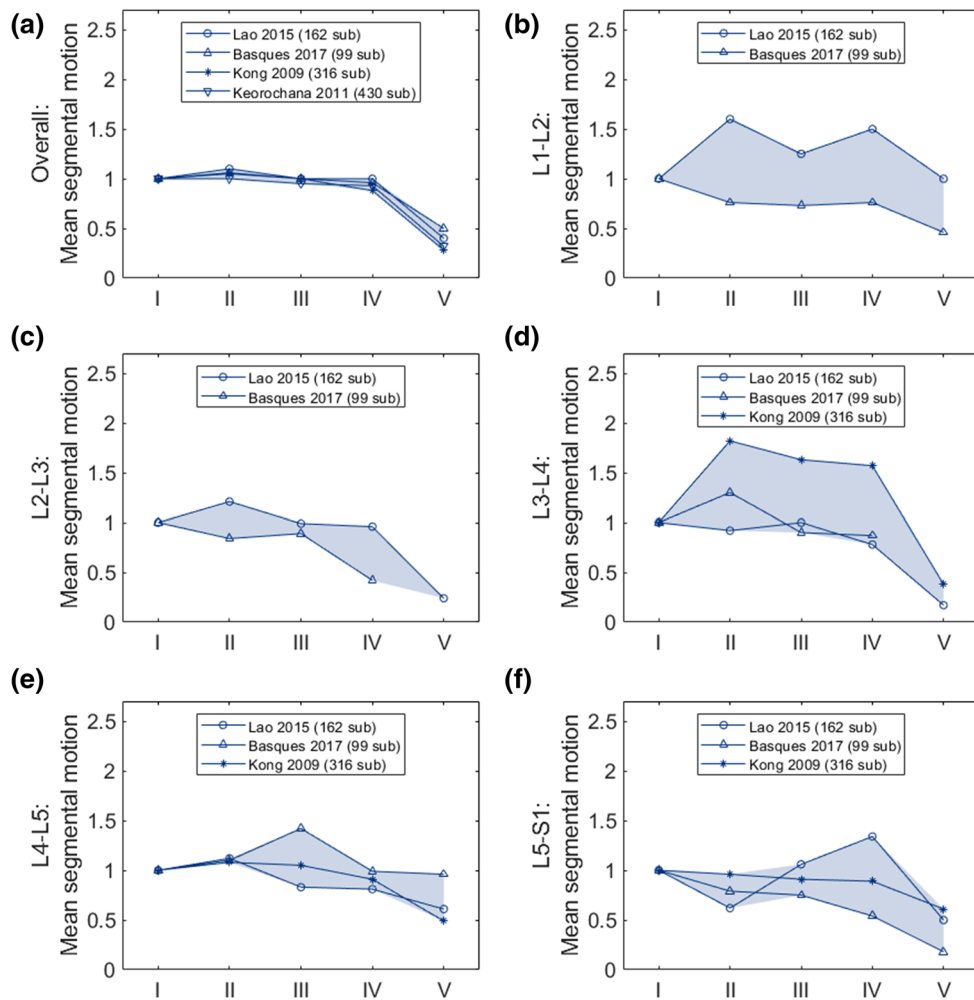
**FIGURE 6.** Angular mean segmental motion during flexion/extension for each lumbar level (b–f) and combined (a), in relation to the Pfirrmann Grading System at the corresponding segment. Data is normalized to the healthy value (grade 1).

mobility drops again at stage 5 to a healthy level. In contrast to the change in angular ROM, translational motion reduction does not take place.

### Center of Rotation

Several authors suggested the COR as a tool to recognize the onset of DD at an early stage.[37,38,103,119,141] However, to the best of our knowledge, no *in vivo* studies were published. The cadaveric study from Seligman *et al.*[119] suggests that DD segments start to move with an erratic motion pattern and thus the COR loses stability and its travel path enlarges during sagittal movement. This phenomenon seems to mainly appear during initial

and intermediate stages of DD and resolves as soon as ankylosing processes come into play. However, cadaveric studies only permit limited insight into *in vivo* behavior.

### Facet Joint Osteoarthritis

The zygapophyseal joints are essential for load transmission,[2] spinal stabilization[1,20,41,121] and strongly influence spinal kinematics.[4] Just as in other types of diarthrodial joints, FJOA leads to the cartilage degradation and therefore focal and diffuse erosions with sclerosis of subchondral bone.[25]
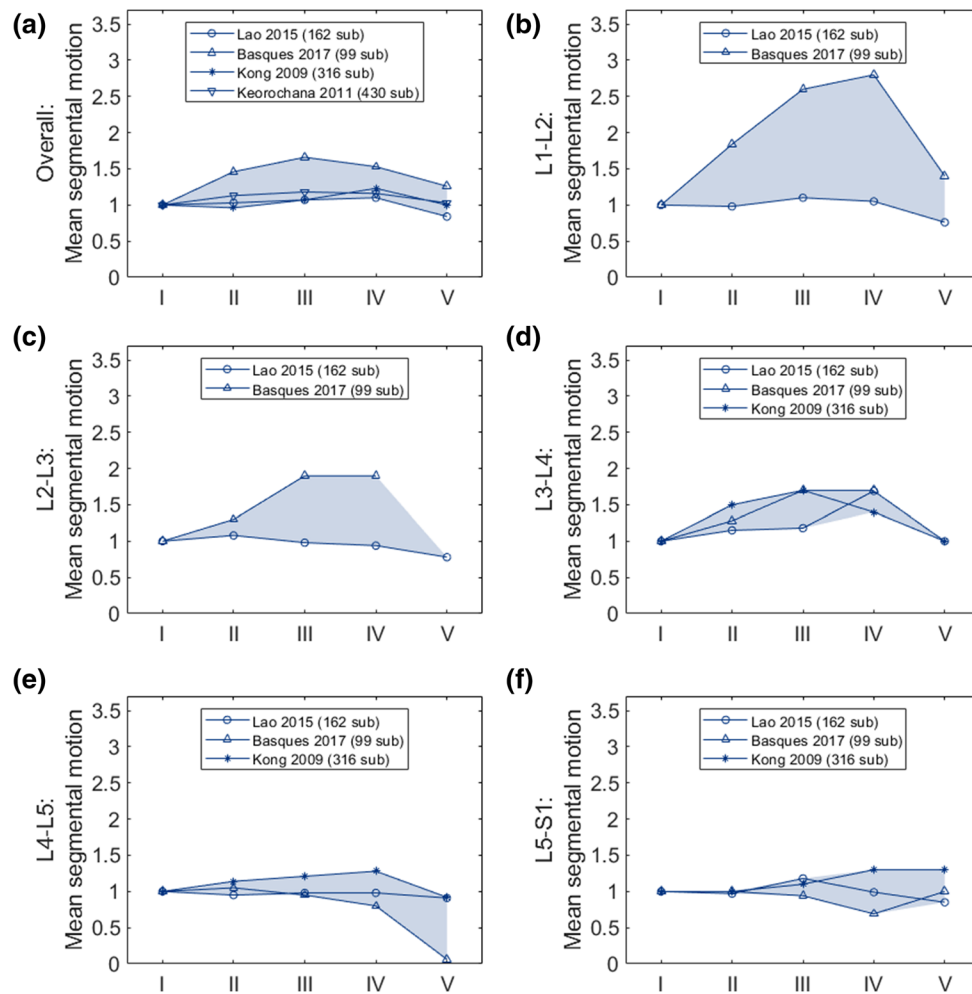
FIGURE 7. Translational mean segmental motion during flexion/extension for each lumbar level (b–f) and combined (a), in relation to the Pfirrmann Grading System at the corresponding segment. Data is normalized to the healthy value (grade 1).

It is etiologically related to aging, injury, gender, facet joint orientation and the spinal level.[54] Besides, strong association with DD was shown,[16,35,41,121] even though the time course of the two diseases is still controversially discussed. Most authors suggest that DD is usually preceding FJOA.[34,35,121,135]

*Range of Motion*

Kong *et al.*[64] investigated the change in angular and translational motion in the sagittal plane in relation to the degree of FJOA, using an MRI based grading system as proposed by Fujiwara.[35] They illustrated that angular mobility seems to monotonically decrease with progressing FJOA (Fig. 8a). On the other hand, translational ROM increased until grade 3 but dropped at the final stage of FJOA (Fig. 8b).

Fujiwara *et al.*[32] divided the pathology of FJOA into three components (subchondral sclerosis, cartilage degeneration, osteophyte formation) and developed a three level grading system to classify each of them (Fig. 8c). Osteophyte formation and cartilage degradation correlate with a ROM pattern proposed by Kirkaldy-Willis: increased angular motion occurs during stage II, which is however reversed to normal ROM values at stage 3. As opposed to this, subchondral sclerosis seems to decrease segmental motion in all planes. A reasonable explanation could be that these are simultaneously ongoing but counteracting processes. Cartilage degeneration loosens the segment, while osteophyte formation and subchondral sclerosis are instantaneously initiated in order to counterbalance segment instability.

*Instability*

Fujiwara *et al.*[33] and Jang[52] investigated the influence of FJOA by defining a translational and angular
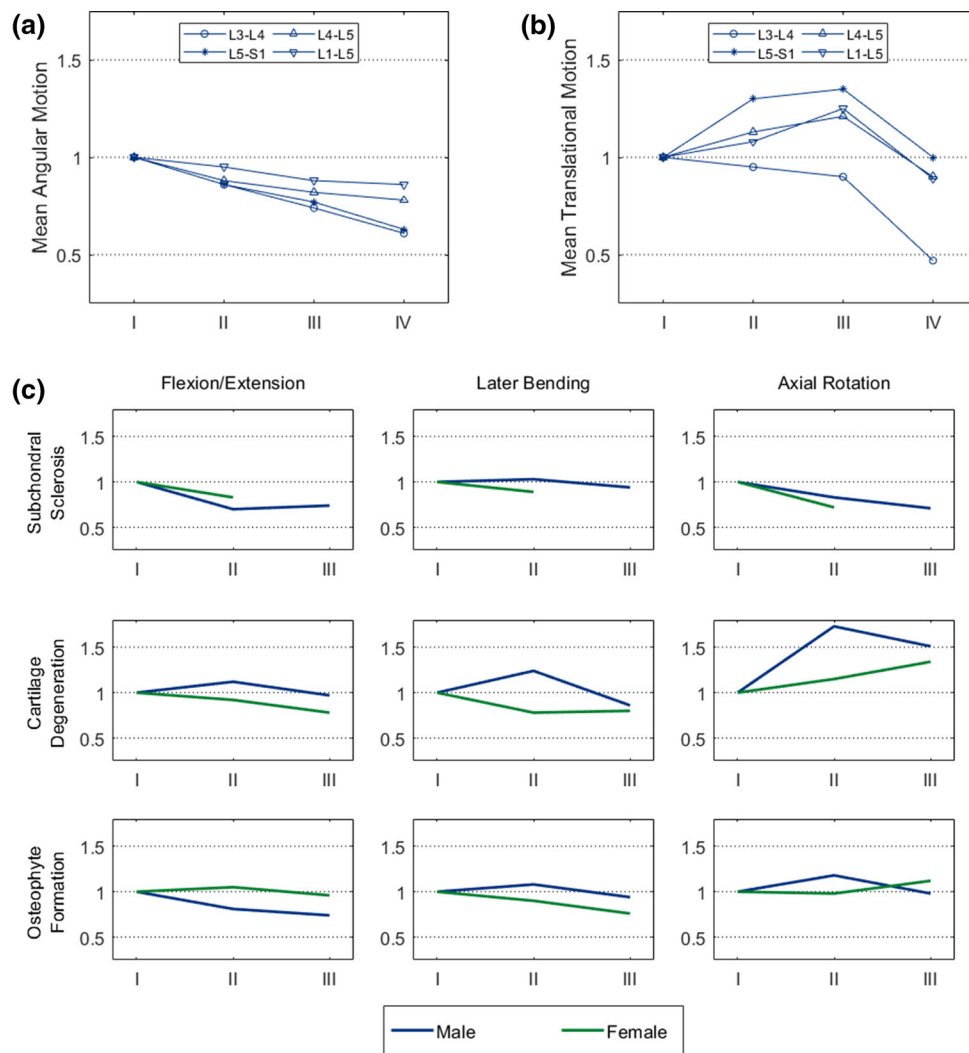
FIGURE 8. Angular motion (a) and translational motion (b) for flexion/extension in relation to the degree of facet joint osteoarthritis according to Fujiwara. (c) Male (blue) and female (green) angular motion in all planes according to the degree of subchondral sclerosis, cartilage degeneration and osteophyte formation. Data is normalized to the healthy value (grade 1).

threshold [max. sagittal ROM: translation > 3 mm, angular motion > 15° (L3–L5)/> 20° (L5–S1)]. Segments exceeding these values were defined as unstable. Fujiwara furthermore divided into anterior and posterior instability. He only found a positive association between FJOA and anterior translatory instability whereas posterior and angular instability were negatively associated. On the other hand, Jang tested instability by complexly correlating DD, FJOA and ligament flavum hypertrophy (LFH). He found angular L4–L5 instability when segments were affected by severe FJOA and LFH. His investigations demonstrate that combined investigations might provide better insight.

*Facet Joint Tropism*

FJT is defined as asymmetry in orientation of the bilateral facets planes. Kong[82] for example determined FJT as an angel difference greater than 7°. To date, it is not clear whether lumbar FJT is congenitally caused or secondary to degeneration. A recent large-scale study within the Asian population however showed that it is age independent, implying that it might be congenital due to a pre-existing phenotype.[115]

Miyazaki *et al.*[84] investigated the influence of facet orientation and FJT on spinal kinematics (Fig. 9). He grouped the facets according to their sagittal angle (angle between sagittal plane and facet plane in transversal the view): "A" are angles smaller than 38.5°
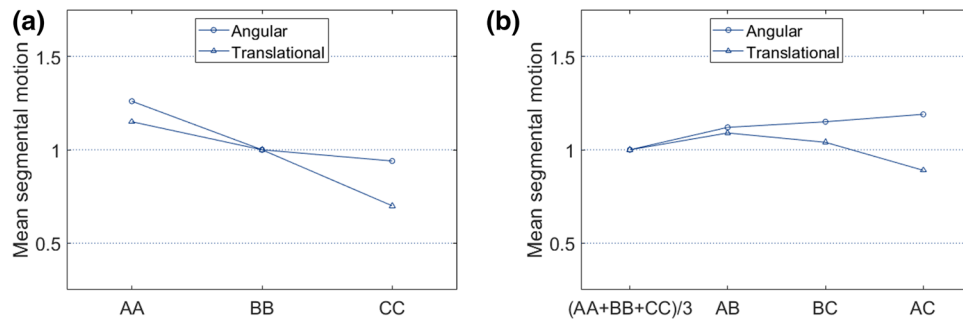
FIGURE 9. Angular and translational motion according to the facet orientation (a) and to the facet tropism (b). "A" are angles smaller than 38.5° (− 0.5 SD), "B" angles from 38.5° to 49° and "C" angles > 49°. In a, data is normalized to the BB value, in b to the average of AA, BB and CC. The illustrated data is from Miyazaka.[84]

(− 0.5 SD), "B" angles from 38.5° to 49° and "C" angles > 49°. He observed that the orientation (AA, BB, CC represent symmetric facet joints) has a significant impact on the translational motion and non-significant but notable influence on the angular motion (Fig. 9a). As it is expected, more sagittal alignment allows increased movement. Similarly, asymmetry of the facet planes played a role (Fig. 9b). However, the data implies that rather the orientation of the facets than the asymmetry is important.

### Spondylolisthesis

SL is the anterior displacement of a vertebra in relation to the natural curve of the spine. Its severity is oftentimes described with the grading system of Meyerding,[78] measuring the percentage of translation of a vertebra relative to its subadjacent vertebra. Grades 1 to 4 represent 0–25, 25–50, 50–75 and 75–100% respectively.

Knutsson et al.[62] first investigated the stability of the malaligned segment in 1944.

In the meantime, the kinematics of SL affected lumbar spines has been frequently studied and a better understanding could be established.

### Range of Motion

Despite the intuitive belief and some early established theories stating that such segments are hypermobile,[62,90] recent literature agrees on normal or even decreased mobility of spondylolisthetic segments.[9,75,79,116,144] These authors could not find any sign of instability, as opposed to Penning[104] and Knuttson,[62] who found hypermobile SL segments.

Figure 10 summarizes all studies providing specific data on the angular motion with and without SL. With

the exception of Phan et al.,[105] who found a slightly increasing ROM for L4–L5 and L5–S1 SL segments, all authors agree on decreasing mobility in SL affected levels.[69,120,141] This loss of motion is attributed to muscle spasm and increased ligament tension due to the slippage between the vertebrae.[102] Interestingly, Takayanagi et al.[127] distinguished between two stages of SL and found an initially increasing and later decreasing ROM, implying that also the effects of SL depend on the severity.

As opposed to this, the ROM for translational movements seems to slightly increase in SL affected segments. However, Takayanagi et al.[127] was the only group who could demonstrate a significant difference.

Whether compensation for increased and decreased motion of SL affected segments occurs in the other segments is not known. Most authors report an overall reduction of the lumber ROM, Yao[147] and Phan[105] showed tendencies towards decreased ROM for the adjacent levels[44] and therefore also for the overall lumbar spine. In line with this, McGregor et al.[77] found a decreased overall lumbar ROM.

Even though most of the presented data does not show any significant changes in ROM, several authors describe erratic motion patterns[79,88,90,91,127] or even backward slipping during flexion.[104,118] Subsequently, we assume that SL alters spinal kinematics in a way that is mainly independent from segmental ROM.

Large-scale studies with dynamical imaging and SL classification according to severity and type (isthmic, degenerative, dysplastic, etc.) are needed for clarification.

### Center of Rotation

Penning et al.[104] investigated the COR of SL segments and noted an increased spread. In contrast, Schneider et al.[22] examined 13 patients and demon-
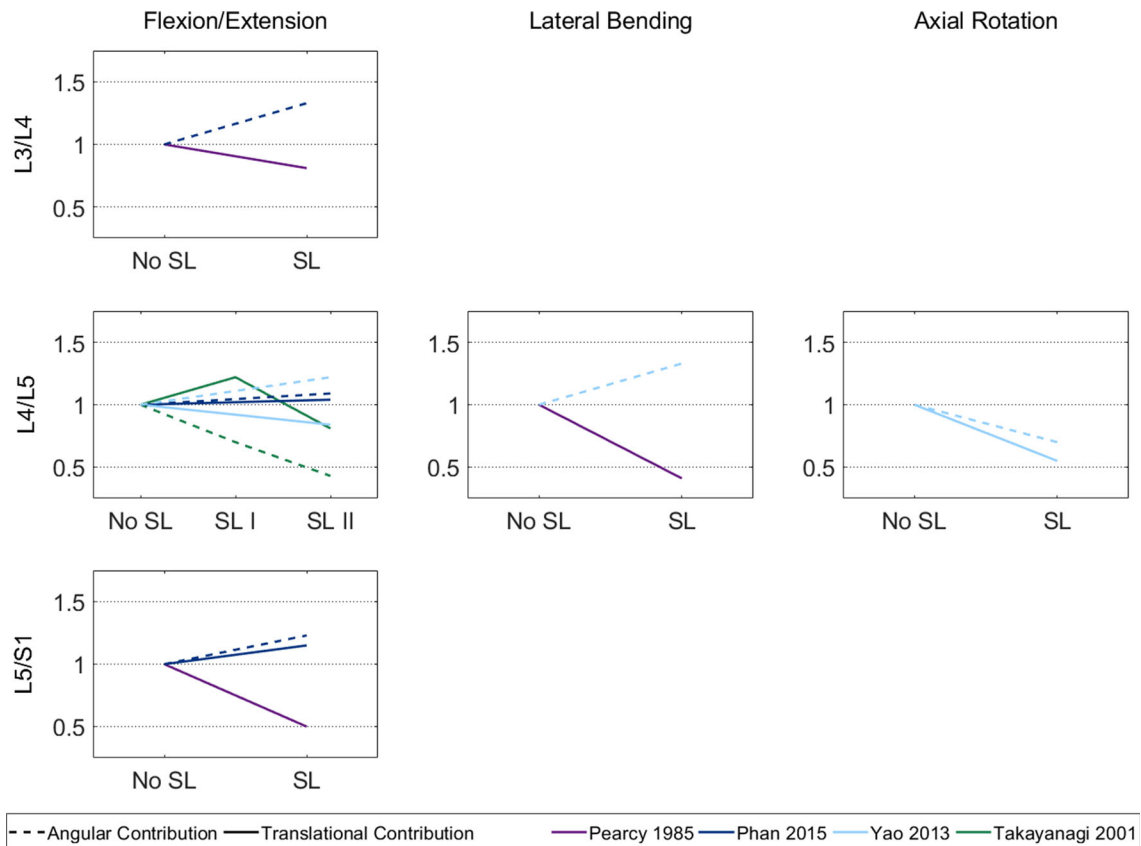
**FIGURE 10.** Angular and translational range of motion with and without spondylolisthesis. Data is normalized to the healthy value (no spondylolisthesis). The illustrated data is from Pearcy,[102] Phan,[105] Yao[147] and Takayanagi.[127]

strated backward slipping of the vertebra during flexion in 6 patients. The COR was found to be located cranially of the disc instead of the healthy location within the subadjacent vertebra.

### Ligament Degeneration

Spinal ligaments significantly contribute to the stability of the spine.[49,50,96] They have been shown to lose tensile strength and elastic modulus with age,[50,59] which is associated with decreased stability. Even though LD was related to lower back pain,[95] only a few authors investigated the relation between LD and altered spine kinematics.

### Range of Motion

Literature is very scarce; we however identified two studies investigating the effects of interspinous ligament (IL) and ligamentum flavum (LF) degeneration on ROM.

*Interspinous Ligament* Figure 11 illustrates the results with quite good agreement between the two groups.[59,64] ROM is slightly increasing during the initial and only drops at the final stage for the more cranial segments. As opposed to this, ROM constantly decreases for L4–L5 during progress of LD, with the exception of stage 3 in the L5–S1 segments, showing a prominent peak of ROM.

No significant impact on translational motion could be found by either of them.

*Ligamentum Flavum* Kong et al.[64] investigated spinal kinematics related to hypertrophy of the LF. Whilst finding no significant difference in angular motion, they demonstrated a significant increase in translational motion in LFH affected spines.

### Paraspinal Muscle Degeneration

PMD has multiple times be shown to accompany lumbar degenerative diseases like DD[122,131] or
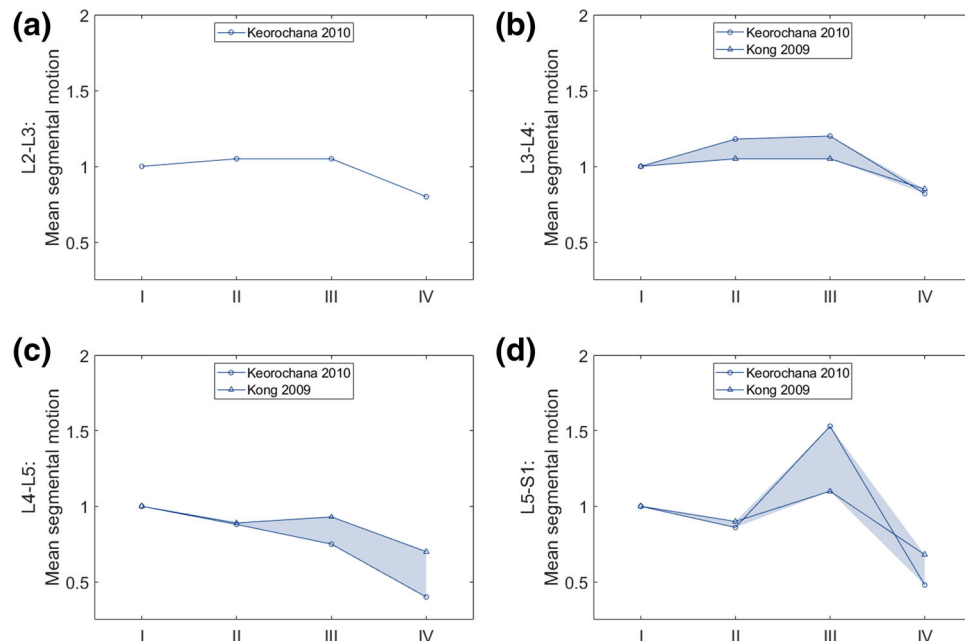
**FIGURE 11. Interspinous ligament degeneration: Angular mean segmental motion in flexion–extension for each lumbar level. Data is normalized to the healthy value (grade 1). Both authors used their own grading system.**

FJOA.[17] Interestingly, it was also observed that animals with degenerative discs, experience a stiffening of the multifidus muscle.[15] It is therefore reasonable to assume that degenerative states of these muscles also influence the kinematics of the spine to some extent. To our knowledge, there is however only one study which investigated the relationship between PMD and the spinal kinematics in the lumbar spine, which is the study of Kong *et al*.[63] He investigated 1575 spine units and split them into a hypermobile (defined as angular ROM > 10° or translational ROM > 3 mm) and a normal cohort. He could show that the presence of fatty muscle infiltration was significantly associated with the presence of excessive angular motion. This effect was however not seen for translational hypermobility. Also at the cervical spine, instability related to PMD could not be observed (except at the C3–C4 level).[51]

## DISCUSSION

This review aims at providing a "state of art" collection on the spinal kinematics under healthy and degenerative conditions. Quite some research has been done since the last review of Panjabi and White,[95,140] which is however still often referred to as state of the art.

Whilst Panjabi and White had to rely on a small selection of mostly *in vitro* studies, an immense range of *in vivo* publications is available today, usually performed with sophisticated measurement techniques. However, despite modern methods, final scientific clarity is widely lacking. Spinal *in vivo* experiments are highly sensitive to experimental parameters and hence, findings are various and sometimes even contradicting. Additionally, with newer methods allowing spatial 3D-tracking of the vertebras, it has become indispensable to agree on very well-defined conventions of how to evaluate results—and to accurately describe them. We therefore would like to emphasize the importance of a precisely defined and described method for future studies. Moreover, common standards and definitions will facilitate comparisons and establishment of reference values.

Besides, sample sizes usually decrease in studies with a complex experimental setup. More sophisticated and therefore accurate methods are time-consuming and expensive, and accordingly suffer from high sample variance due to a small patient population. On the other hand, simpler methods can be applied to a larger population, but lose quality due to the inaccuracy of the technique. However, immense technical possibilities are given and the diversity of experimental parameters is well known by now. The door is therefore wide open for large-scale studies with great

potential to provide clarification of the pressing questions.

We initially asked the question of whether it is possible to define healthy kinematic behavior and clearly distinguish it from the motion pattern of a degenerated spine. In conclusion, the answer has to be: not yet. As summarized in the section on degenerated spines, many diseases are associated with significant differences in kinematical behavior. However, due to high individuality, healthy and pathological ranges largely overlap and a clear distinction is therefore not yet feasible.

An interesting finding is that increasing stiffness of a segment during DD does not immediately produce decreasing motion. ROM is only reduced at the final stage, when severe ankylosing processes come into play. We thus conclude that mechanical stiffness is compensated by increased muscular activity to enable end-positions of certain movements. Cadaveric and also finite element studies, both neglecting muscular components, therefore have very limited power to simulate *in vivo* behavior.

What remains unclear is to what extent ankylosing processes are involved in restabilization of a segment as described by Kirkaldy-Willis.[61] Many figures in this review follow the proposed pattern, suggesting that such mechanisms indeed exists. On the other hand, examples of a monotonically decreasing motion pattern challenge the Kirkaldy-Willis theory and therefore call for more research.

Another essential point to keep in mind is to not only look at degenerative changes as an isolated disease but to incorporate all of the simultaneously ongoing processes. Jang et al.[52] could show correlations between kinematical changes and combinations of degenerative diseases. Broadening our perspective accordingly could lead to a better understanding in the field.

We also like to emphasize that more research is required in some of the topics. Particularly, this is LD and PMD. Since, the impact of these degenerative changes are not understood well enough, they are also not involved in the clinical diagnostics nowadays.

In conclusion it can be said that research still is in the middle of investigating extremely complex processes. Hence, direct clinical value in order to use kinematical parameters as a tool to diagnose or to make decisions on surgical interventions is very limited to date. However, we are confident that this will change in the future.

As a limitation of this review, we would like to point out the possibility that some publications were missed by the choice of the key words. We tried to select the keywords very general and also included a subsequent reference search in order to find most of the studies. However, medical and biomechanical terms for the same topics can strongly vary and therefore, we cannot completely exclude that some studies were missed.

## APPENDIX

### *(Angular/Translational) Segmental Motion Contribution*

See Fig. 12 and Table 1.



**MEDLINE SEARCH**:

Keyword: lumbar spine[Title/Abstract] AND (kinematics[Title/Abstract] OR motion[Title/Abstract] OR flexion[Title/Abstract] OR extension[Title/Abstract] OR axial rotation[Title/Abstract] OR lateral bending[Title/Abstract] )

**INCLUSION CRITERIA**:          **2992 Studies**

Time range:      1970–2016
Method:          in-vivo, all measurement techniques
Data:            Angular: physiological, intervertebral angle change (° or %)
                 Translation: physiological, translational change (° or %)
                 Exclusion: Pearcy [101] (due to too low value resolution) and Dvorak [24] for translation (incomparable translation measurement method)

**31 Studies**

**REFERENCE SEARCH**:     **5 Studies**       **FINAL LITERATURE**:
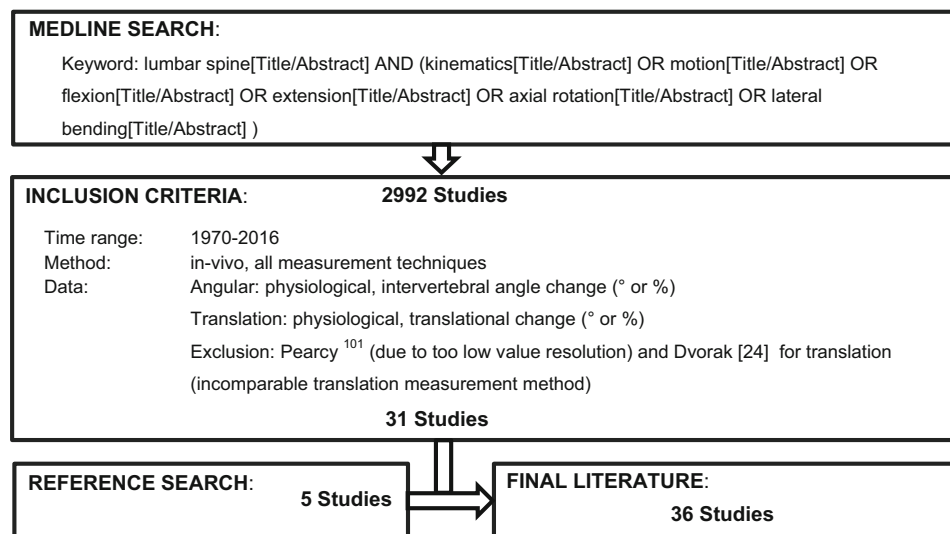                                               **36 Studies**

FIGURE 12. Search strategy for studies on segmental motion contribution.

**TABLE 1. Results of the search for literature on segmental motion contribution.**

| Methods | Subject numbers | Years | Authors |
|---|---|---|---|
| CT + dual-videofluoroscopy, dynamic, *in vivo*, FE | 14 | 2015 | Aiyangar[5] |
| CT + dual-videofluoroscopy, dynamic, *in vivo*, FE | 8 | 2014 | Aiyangar[7] |
| MRI + dual-videofluoroscopy, dynamic, *in vivo*, FE | 10 | 2014 | Wu[145] |
| Fluoroscopy, incremental, *in vivo*, LB | 12 | 2013 | Hashemirad[45] |
| MRI + dual-videofluoroscopy, *in vivo*, AR | 8 | 2013 | Shin[123] |
| MRI, initial–final-position, *in vivo*, FE | 100 | 2012 | Tan[129] |
| MRI, initial–final-position, *in vivo*, FE | 294 | 2011 | Keorochana[58] |
| MRI + dual fluoroscopy, incremental, *in vivo*, FE + LB + AR | 10 | 2011 | Passias[98] |
| Videofluoroscopy, dynamic, *in vivo*, FE | 15 | 2009 | Ahmadi[3] |
| MRI + dual fluoroscopy, incremental, *in vivo*, FE + LB + AR | 8 | 2009 | Li[71] |
| Kirshner wires, dynamic, *in vivo*, FE + LB + AR | 10 | 2008 | Rozumalski[112] |
| CT + dual-videofluoroscopy, dynamic, *in vitro/vivo*, FE | 1 + 1 | 2008 | Wang[138] |
| MRI, incremental position, *in vivo*, AR | 10 | 2007 | Fujii[31] |
| MRI, initial–final position, *in vivo*, FE (horizontal) | 20 | 2007 | Kulig[66] |
| CT, incremental, *in vivo*, AR | 10 | 2007 | Ochia[87] |
| Videofluoroscopy, dynamic, *in vivo*, FE | 20 | 2007 | Teyhen[132] |
| CT, initial–final position, *in vivo*, AR | 16 | 2006 | Blankenbaker[13] |
| CT, incremental, *in vivo*, AR | 16 | 2006 | Ochia[86] |
| Videofluoroscopy, dynamic, *in vivo*, FE | 30 | 2006 | Wong[142] |
| Videofluoroscopy, dynamic, *in vivo*, FE | 100 | 2004 | Wong[143] |
| MRI, initial–final-position, *in vivo*, AR | 5 | 2002 | Haughton[46] |
| Videofluoroscopy, dynamic, *in vivo*, FE | 30 | 2002 | Lee[70] |
| Cineradiographic, dynamic, *in vivo*, FE | 20 | 2001 | Takayanagi[127] |
| Cineradiographic, dynamic, *in vivo*, FE | 10 | 2000 | Harada[43] |
| AP X-ray, initial–final-position, *in vivo*, FE | 90 | 2000 | Miyasaka[83] |
| Videofluoroscopy, dynamic, *in vivo*, FE | 13 | 1998 | Okawa[89] |
| AP X-ray, initial–final-position, *in vivo*, FE | 37–59 | 1996 | Frobin[29] |
| Cineradiographic, dynamic, *in vivo*, FE | 8 | 1996 | Kanayama[56] |
| AP X-ray, initial–final-position, *in vivo*, FE | 56 | 1992 | Tallroth[128] |
| AP X-ray, initial–final-position, *in vivo*, FE + LB | 41 | 1991 | Dvorak[23] |
| AP X-ray, initial–final-position, *in vivo*, FE | 101 | 1991 | Dvorak[24] |
| Biplanar radiography, incremental, *in vivo*, FE | 40 | 1990 | Boden[14] |
| Biplanar radiography, incremental, *in vivo*, LB | 50 | 1990 | Mimura[80] |
| AP X-ray, initial–final-position, *in vivo*, FE | 59 | 1988 | Hayes[48] |
| Biplanar radiography, initial–final-position, *in vivo*, FE + LB + AR | 16 | 1988 | Plamondon[106] |
| Biplanar radiography, initial–final-position, *in vivo*, FE + LB + AR | 11 | 1985 | Pearcy[100] |

*Maximal Range of Motion*                                    *Coupling*

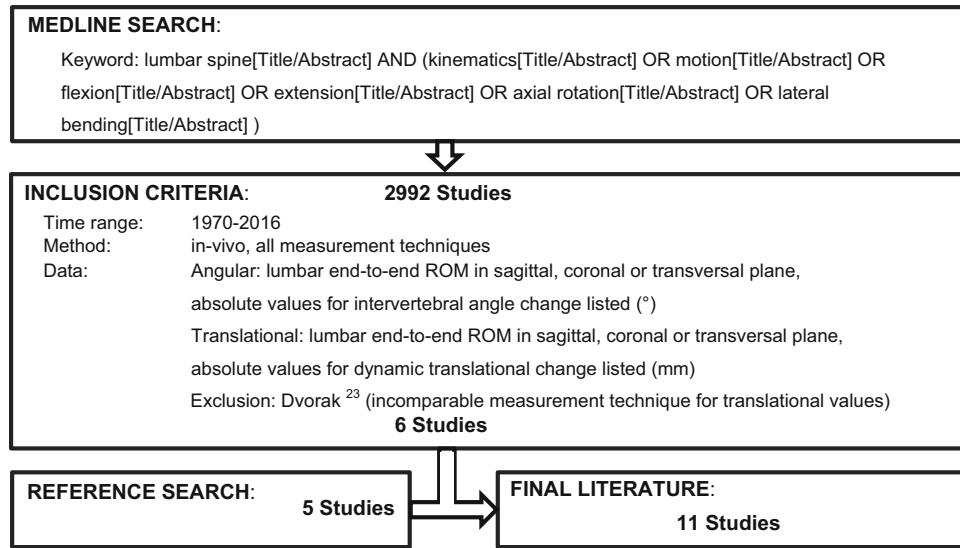See Fig. 13 and Table 2.                              See Fig. 14 and Table 3.

**MEDLINE SEARCH**:

    Keyword: lumbar spine[Title/Abstract] AND (kinematics[Title/Abstract] OR motion[Title/Abstract] OR

    flexion[Title/Abstract] OR extension[Title/Abstract] OR axial rotation[Title/Abstract] OR lateral

    bending[Title/Abstract] )

⇩

**INCLUSION CRITERIA**:                    **2992 Studies**

| Time range: | 1970-2016 |
| Method: | in-vivo, all measurement techniques |
| Data: | Angular: lumbar end-to-end ROM in sagittal, coronal or transversal plane, |

    absolute values for intervertebral angle change listed (°)

    Translational: lumbar end-to-end ROM in sagittal, coronal or transversal plane,

    absolute values for dynamic translational change listed (mm)

    Exclusion: Dvorak [23] (incomparable measurement technique for translational values)

**6 Studies**

| **REFERENCE SEARCH**: | 5 Studies | → | **FINAL LITERATURE**: |
| | | | **11 Studies** |

FIGURE 13.  Search strategy for studies on maximal range of motion.

TABLE 2.  Results of the search for literature on maximal range of motion.

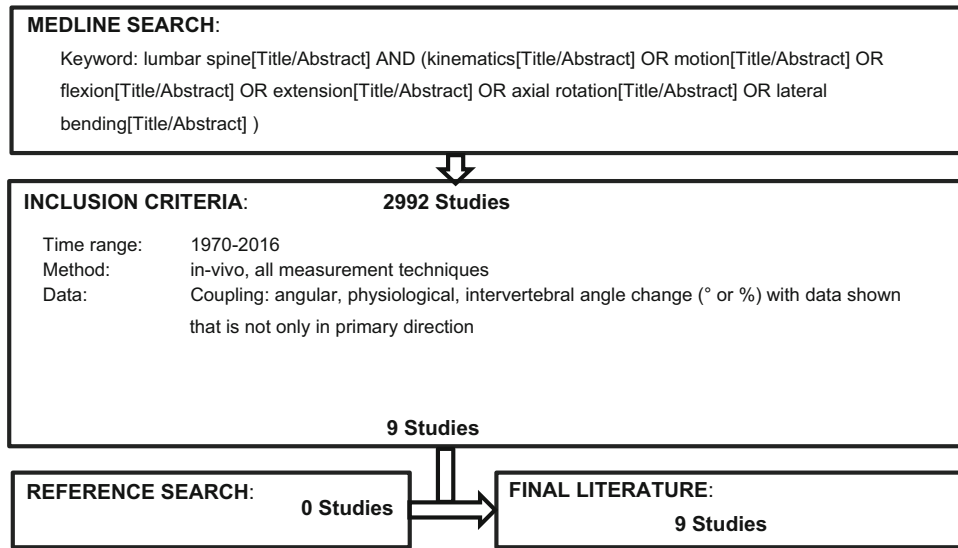| Methods | Subject numbers | Years | Authors |
|---|---|---|---|
| MRI + dual-Videofluoroscopy, *in vivo*, AR | 8 | 2013 | Shin[123] |
| MRI + dual fluoroscopy, incremental, *in vivo*, FE + LB + AR | 8 | 2009 | Li[71] |
| Kirshner wires, dynamic, *in vivo*, FE + LB + AR | 10 | 2008 | Rozumalski[112] |
| AP X-ray, initial–final-position, *in vivo*, FE | 37–59 | 1996 | Frobin[29] |
| AP X-ray, initial–final-position, *in vivo*, FE | 56 | 1992 | Tallroth[128] |
| AP X-ray, initial–final-position, *in vivo*, FE + LB | 41 | 1991 | Dvorak[23] |
| AP X-ray, initial–final-position, *in vivo*, FE | 101 | 1991 | Dvorak[24] |
| Biplanar radiography, incremental, *in vivo*, FE | 40 | 1990 | Boden[14] |
| Biplanar radiography, incremental, *in vivo*, LB | 50 | 1990 | Mimura[80] |
| AP X-ray, initial–final-position, *in vivo*, FE | 59 | 1989 | Hayes[48] |
| Biplanar radiography, initial–final-position, *in vivo*, FE + LB + AR | 11 | 1985 | Pearcy[100] |

*Center of Rotation*

See Fig. 15 and Table 4.

**MEDLINE SEARCH**:

Keyword: lumbar spine[Title/Abstract] AND (kinematics[Title/Abstract] OR motion[Title/Abstract] OR flexion[Title/Abstract] OR extension[Title/Abstract] OR axial rotation[Title/Abstract] OR lateral bending[Title/Abstract] )

**INCLUSION CRITERIA**: **2992 Studies**

Time range:    1970-2016
Method:    in-vivo, all measurement techniques
Data:    Coupling: angular, physiological, intervertebral angle change (° or %) with data shown that is not only in primary direction

**9 Studies**

**REFERENCE SEARCH**: **0 Studies**

**FINAL LITERATURE**: **9 Studies**

FIGURE 14.   Search strategy for studies on coupled motion.

TABLE 3.   Results of the search for literature on coupled motion.

| Methods | Subject numbers | Years | Authors |
|---|---|---|---|
| CT + dual-videofluoroscopy, dynamic, *in vivo*, FE | 8 | 2014 | Aiyangar[7] |
| MRI + dual-videofluoroscopy, dynamic, *in vivo*, FE | 10 | 2014 | Wu[145] |
| MRI + dual-videofluoroscopy, *in vivo*, AR | 8 | 2013 | Shin[123] |
| MRI + dual fluoroscopy, incremental, *in vivo*, FE + LB + AR | 8 | 2009 | Li[71] |
| CT + dual-videofluoroscopy, dynamic, *in vitro/vivo*, FE | 1 + 1 | 2008 | Wang[138] |
| Kirshner wires, dynamic, *in vivo*, FE + LB + AR | 10 | 2008 | Rozumalski[112] |
| MRI, incremental position, *in vivo*, AR | 10 | 2007 | Fujii[31] |
| CT, incremental, *in vivo*, AR | 10 | 2007 | Ochia[87] |
| CT, incremental, *in vivo*, AR | 16 | 2006 | Ochia[86] |

**MEDLINE SEARCH**:

Keyword: lumbar spine[Title/Abstract] AND (rotation[Title/Abstract] OR axis[Title/Abstract])

**INCLUSION CRITERIA**: **1054 Studies**

Time range:    1970-2016
Method:    in-vivo, in vitro (for LB and AR), all measurement techniques
Data:    physiological COR, IAR or HAM visualized, lumbar spine

**10 Studies**

**REFERENCE SEARCH**: **5 Studies**

**FINAL LITERATURE**: **15 Studies**
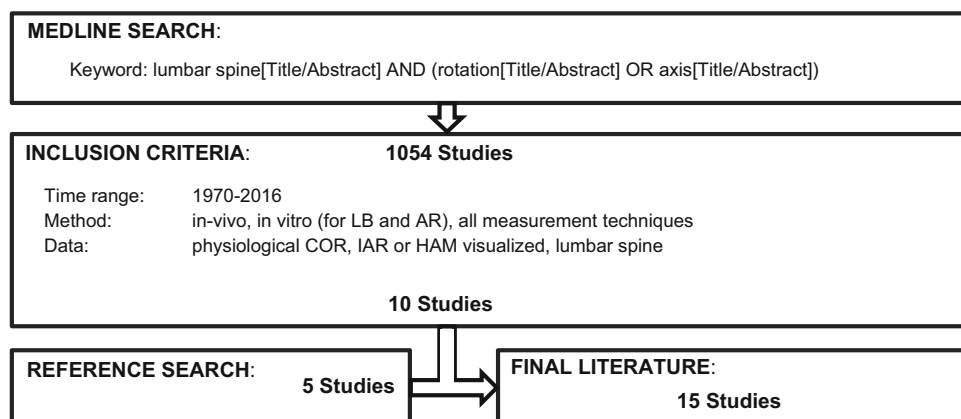
FIGURE 15.   Search strategy for studies on the center of rotation.

*Phase Lag*

See Fig. 16 and Table 5.

TABLE 4. Results of the search for literature on the center of rotation.

| Methods | Subject numbers | Years | Authors |
|---|---|---|---|
| CT + dual-videofluoroscopy, dynamic, *in vivo*, FE | 14 | 2017 | Aiyangar[6] |
| Displacement sensors, dynamic, *in vitro*, AR | 1 | 2010 | Wachowski[137] |
| MRI + fluoroscopic, *in vivo*, FE + AR | 10 | 2010 | Xia[146] |
| Planar X-ray, incremental, *in vitro*, FE | 12 | 2006 | Rousseau[111] |
| Displacement sensors, dynamic, *in vitro*, AR | 1 | 2003 | Mansour[74] |
| Planar X-ray, initial–final-position, *in vivo*, FE | 70 | 2002 | Sakamaki[113] |
| Photograph, initial–final-position, *in vitro*, FE | 10 | 1991 | Haher[40] |
| Planar X-ray, initial–final-position, *in vivo*, FE | 61 | 1990 | Yoshioka[148] |
| Planar X-ray, initial–final-position, *in vivo*, FE | 10 | 1988 | Pearcy[101] |
| Moiré fringes, incremental, *in vitro*, FE | 47 | 1986 | Gertzbein[37] |
| Planar X-ray, incremental, *in vivo*, FE | 44 | 1986 | Ogston[88] |
| Moiré fringes, incremental, *in vitro*, FE | 28 | 1985 | Gertzbein[38] |
| Planar X-ray, incremental, *in vitro*, FE | 47 | 1984 | Seligman[119] |
| Biplanar X-ray, initial–final-position, *in vitro/vivo*, FE + LB + AR | Not stated | 1977 | Pope[107] |
| Photograph, initial–final-position, *in vitro*, AR | 12 | 1971 | Cossette[18] |

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┌─────────────────────────────────────────────────────────────────────────┐
│ MEDLINE SEARCH:                                                           │
│     Keyword: lumbar spine [Title/Abstract] AND (kinematics [Title/Abstract] OR motion [Title/Abstract] OR │
│     flexion [Title/Abstract] OR extension [Title/Abstract] OR axial rotation [Title/Abstract] OR lateral bending │
│     [Title/Abstract] OR phase lag [Title/Abstract])                       │
└─────────────────────────────────────────────────────────────────────────┘
                                    ⇩
┌─────────────────────────────────────────────────────────────────────────┐
│ INCLUSION CRITERIA:              2992 Studies                             │
│                                                                           │
│     Time range:     1970-2016                                             │
│     Method: in-vivo, all measurement techniques                           │
│     Data:           Dynamic intervertebral angle measurement (° or %)     │
│                                                                           │
│                          8 Studies                                        │
└─────────────────────────────────────────────────────────────────────────┘
┌──────────────────────────────┐      ┌────────────────────────────────┐
│ REFERENCE SEARCH:            │─────▶│ FINAL LITERATURE:              │
│              1 Study         │      │              9 Studies         │
└──────────────────────────────┘      └────────────────────────────────┘
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FIGURE 16. Search strategy for studies on phase lag.

TABLE 5. Results of the search for literature on phase lag.

| Methods | Subject numbers | Years | Authors |
|---|---|---|---|
| CT + dual-videofluoroscopy, dynamic, *in vivo*, FE | 8 | 2014 | Aiyangar[7] |
| Videofluoroscopy, dynamic, *in vivo*, FE | 20 | 2007 | Teyhen[132] |
| Videofluoroscopy, dynamic, *in vivo*, FE | 30 | 2006 | Wong[142] |
| Videofluoroscopy, dynamic, *in vivo*, FE | 100 | 2004 | Wong[143] |
| Videofluoroscopy, dynamic, *in vivo*, FE | 30 | 2002 | Lee[70] |
| Cineradiographic, dynamic, *in vivo*, FE | 10 | 2000 | Harada[43] |
| Cineradiographic, dynamic, *in vivo*, FE | 20 | 2000 | Takayanagi[127] |
| Fluoroscopy, dynamic, *in vivo* (path.), FE | 21 | 1998 | Okawa[89] |
| Cineradiographic, dynamic, *in vivo*, FE | 8 | 1996 | Kanayama[56] |

*Disc Degeneration*

*Mechanical Stiffness*

See Fig. 17 and Table 6.

*Segmental and overall ROM*

See Fig. 18 and Tables 7 and 8.
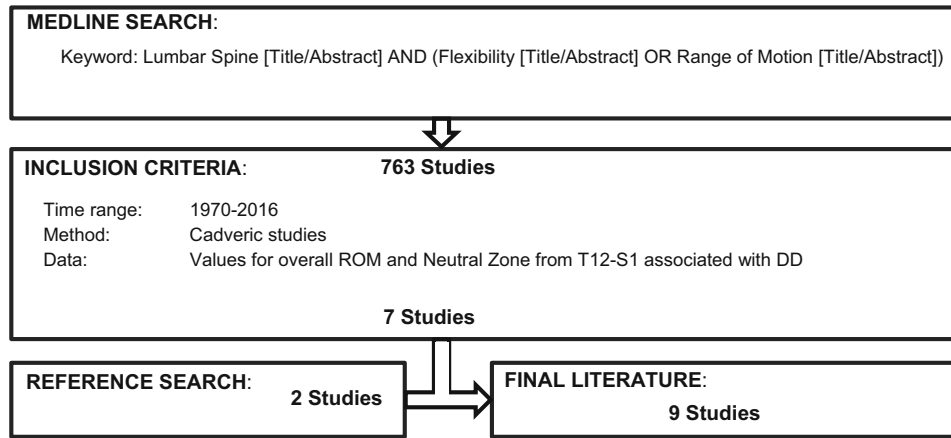


FIGURE 17.   Search strategy for studies on disc degeneration and mechanical stiffness of the tissue.

TABLE 6.   Results of the search for literature on disc degeneration and mechanical stiffness of the tissue.

| Methods | Specimen numbers | Years | Authors |
|---|---|---|---|
| *In vitro*, ROM + stiffness + hysteresis measurement, FE + LB + AR | 54 | 2016 | Muriuki[85] |
| Review | – | 2014 | Gakbzsera[36] |
| *In vitro*, ROM + stiffness + hysteresis measurement, FE + LB + AR | 21 | 2013 | Zirbel[150] |
| *In vitro*, ROM + NZ measurement, FE + LB + AR | 203 | 2011 | Kettler[60] |
| *In vitro*, ROM + NZ + NZR measurement, FE + LB + AR | 18 | 2008 | Quint[109] |
| *In vitro*, ROM measurement, FE + LB + AR | 114 | 2001 | Tanaka[130] |
| *In vitro*, ROM measurement, FE + LB + AR | 110 | 2000 | Fujiwara[32] |
| *In vitro*, ROM measurement, AR | 36 | 2000 | Krismer[65] |
| *In vitro*, ROM + NZ measurement, FE + LB + AR | 24 | 1996 | Oxland[92] |
| *In vitro*, ROM + NZ measurement, FE + LB + AR | 47 | 1993 | Mimura[81] |
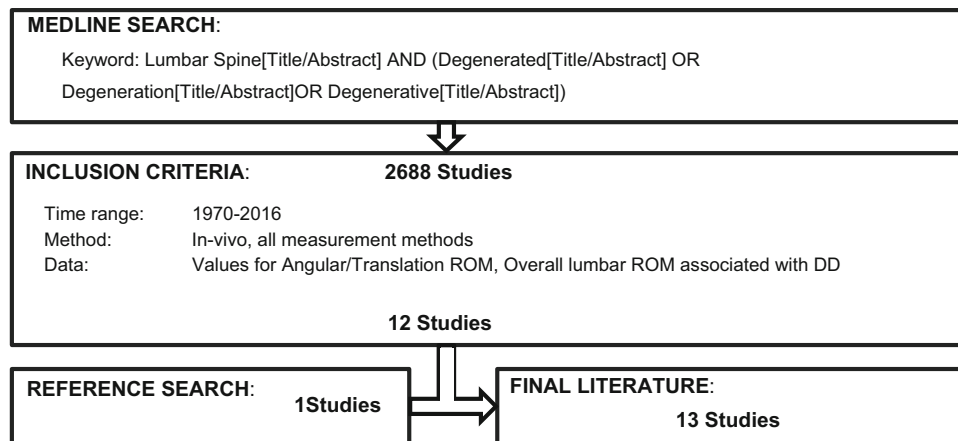


FIGURE 18.   Search strategy for studies on spinal ROM and disc degeneration.

**TABLE 7. Results of the search for literature on spinal ROM and disc degeneration.**

| Methods | Subject numbers | Years | Authors |
|---|---|---|---|
| *In vivo*, angular/translational motion with DD, FE | 99 | 2016 | Basques[10] |
| *In vivo*, angular/translational motion with DD, FE (no grading) | 450 | 2015 | Hayashi[47] |
| *In vivo*, angular/translational motion with DD, FE | 162 | 2014 | Lao[67] |
| *In vivo*, motion compensation with DD, FE | 259 | 2014 | Lee[68] |
| *In vivo*, angular/translational motion with DD, FE | 430 | 2011 | Keorochana[58] |
| *In vivo*, angular/translational motion with DD, FE + LB + AR (no grading) | 10 | 2011 | Li[72] |
| *In vivo*, angular/translational motion with DD, FE | 316 | 2009 | Kong[64] |
| *In vivo*, overall lumbar motion with DD, FE + LB | 112 | 2007 | Quack[108] |
| *In vivo*, supine vs. sitting with DD, FE | 30 | 2006 | Karadimas[57] |
| *In vivo*, overall lumbar motion with DD, FE | 57 | 1998 | McGregor[76] |
| *In vivo*, overall lumbar motion with DD, FE | 100 | 1988 | Kambin[55] |
| *In vivo*, angular/translational motion with DD, FE + LB + AR (no grading) | 78 | 1984 | Frymoyer[30] |
| *In vivo*, overall lumbar motion with DD, FE | 604 | 1976 | Torgerson[134] |

**TABLE 8. Dotted lines → neutral zone?).**
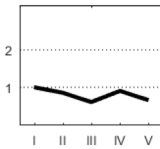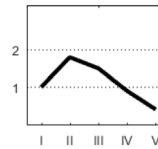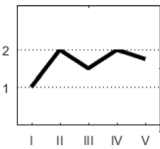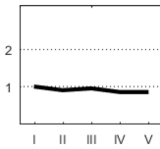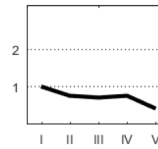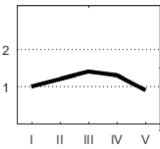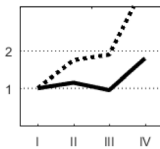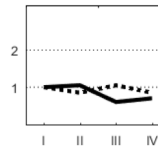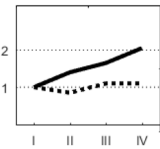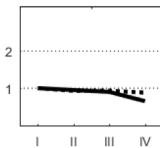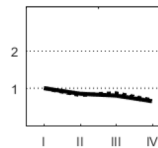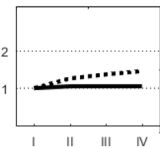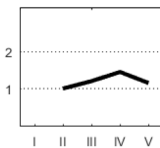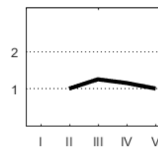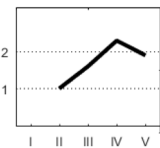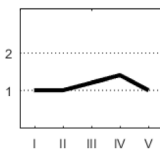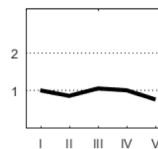
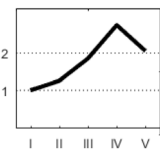| Authors | Sample size | Classification system | Moment (ax. load) | ROM flexion–extension | ROM lateral bending | ROM axial rotation |
|---|---|---|---|---|---|---|
| Zirbel[150] | 21 | Thompson | 7.5 Nm (440 N) | | | |
| Muriuki[85] | 270 | Pfirrmann | 8 Nm (400 N) | | | |
| Oxland[92] | 24 | Vernon-Roberts | 10 Nm (200 N) | | | |
| Kettler[60] | 203 | Wilke | 7.5 Nm (0 N) | | | |
| Fujiwara[32] | 110 | Thompson | 6.6 Nm (0 N) | | | |
| Tanaka[130] (T12–L4) | 81 | Thompson | 5.7 Nm (0 N) | | | |

**TABLE 8.  continued.**
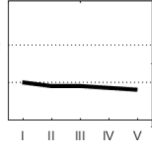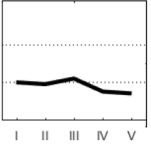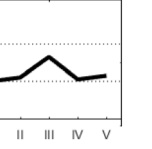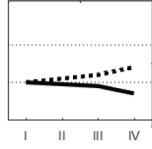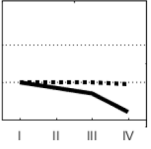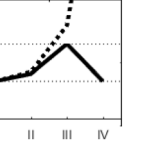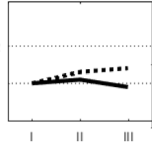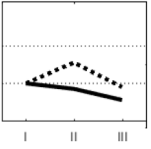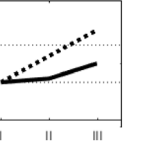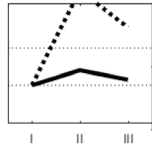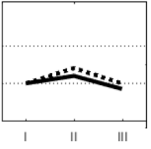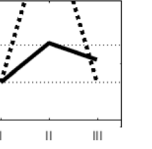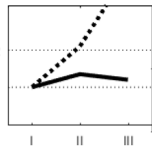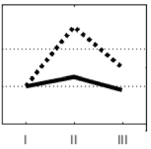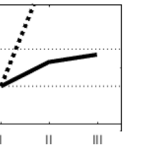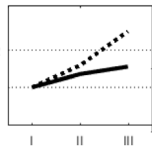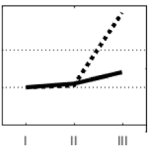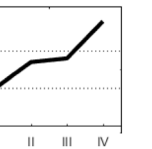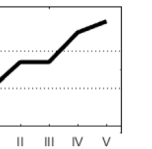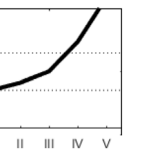
| Authors | Sample size | Classification system | Moment (ax. load) | ROM flexion–extension | ROM lateral bending | ROM axial rotation |
|---|---|---|---|---|---|---|
| Tanaka[130] (L4–S1) | 33 | Thompson | 5.7 Nm (0 N) | *[chart]* | *[chart]* | *[chart]* |
| Mimura[81] | 60 | Mimura radiographic | 10 Nm (0 N) | *[chart]* | *[chart]* | *[chart]* |
|  |  | Mimura macroscopic |  | *[chart]* | *[chart]* | *[chart]* |
| Quint[109] | 18 | Mimura radiographic | 7.5 Nm (0 N) | *[chart]* | *[chart]* | *[chart]* |
|  |  | Nachemson |  | *[chart]* | *[chart]* | *[chart]* |
|  |  | Vernon-Roberts |  | *[chart]* | *[chart]* | *[chart]* |
| Krismer[65] | 36 | Nachemson | 8.5 Nm (0 N) |  |  | *[chart]* |
|  |  | Thompson |  |  |  | *[chart]* |
|  |  | Adams |  |  |  | *[chart]* |

**TABLE 8. continued.**

| Authors | Sample size | Classification system | Moment (ax. load) | ROM flexion–extension | ROM lateral bending | ROM axial rotation |
|---|---|---|---|---|---|---|
| | | Mimura radiographic | | | |  |



FIGURE 19. Search strategy for studies on spinal ROM and facet joint osteoarthrosis.

**TABLE 9. Results of the search for literature on spinal ROM and facet joint osteoarthrosis.**

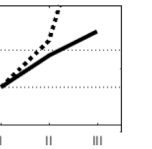| Methods | Subject numbers | Years | Authors |
|---|---|---|---|
| *In vivo*, angular/translational motion with FJOA (instability criterion), FE | 309 | 2009 | Jang[52] |
| *In vivo*, angular/translational motion with FJOA, FE + LB + AR | 316 | 2009 | Kong[64] |
| *In vivo*, angular/translational motion with FJOA, FE + LB + AR | 110 | 2000 | Fujiwara[32] |
| *In vivo*, angular/translational motion with FJOA (instability criterion), FE | 70 | 2000 | Fujiwara[33] |

*Facet Joint Osteoarthrosis*

See Fig. 19 and Table 9.

*Facet Joint Tropism*

See Fig. 20 and Table 10.

*Spondylolisthesis*

See Fig. 21 and Table 11.

**MEDLINE SEARCH**:

    Keyword: Lumbar Spine[Title/Abstract] AND Tropism [Title/Abstract]

**INCLUSION CRITERIA**:        **38 Studies**

Time range:    1970-2016
Method:    In-vivo, all measurement methods
Data:    Values for Angular/Translation ROM, Overall lumbar ROM associated with FJT

**1 Study**

**REFERENCE SEARCH**:    **0 Studies**    **FINAL LITERATURE**:    **1 Study**

**FIGURE 20.** Search strategy for studies on spinal ROM and facet joint tropism.
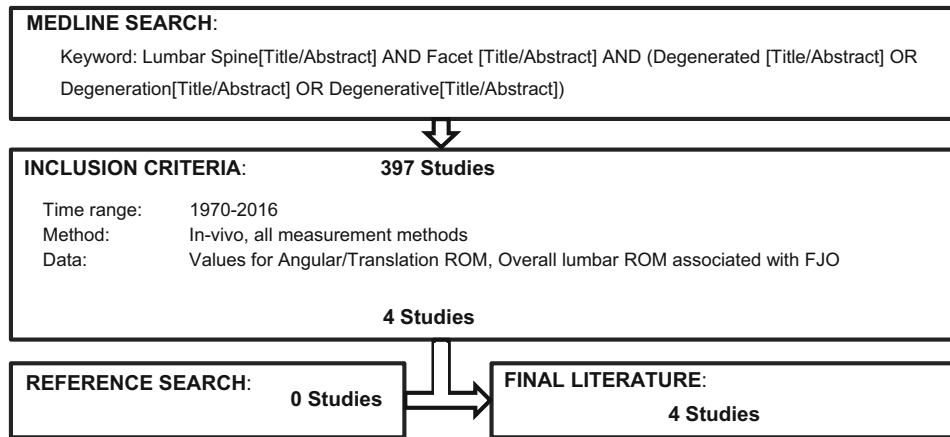
**TABLE 10.** Results of the search for literature on spinal ROM and facet joint tropism.

| Method | Subject number | Year | Author |
|---|---|---|---|
| *In vivo*, angular/translational motion with FJT, FE | 305 | 2000 | Fujiwara[32] |

**MEDLINE SEARCH**:

    Keyword: Lumbar Spine[Title/Abstract] AND Spondylolisthesis [Title/Abstract]

**INCLUSION CRITERIA**:        **283 Studies**

Time range:    1970-2016
Method:    In-vivo, all measurement methods
Data:    Values for Angular/Translation ROM, Overall lumbar ROM associated with SL

**13 Studies**

**REFERENCE SEARCH**:    **4 Studies**    **FINAL LITERATURE**:    **17 Studies**

**FIGURE 21.** Search strategy for studies on spinal ROM and spondylolisthesis.

*Ligament Degeneration*

See Fig. 22 and Table 12.

**TABLE 11. Results of the search for literature on spinal ROM and spondylolisthesis.**

| Methods | Subject numbers | Years | Authors |
|---|---|---|---|
| *In vivo*, angular/translational motion with SL, FE | 112 | 2015 | Phan[105] |
| *In vivo*, instability with SL, FE | 9 | 2013 | Miao[79] |
| *In vivo*, angular/translational motion with SL, FE + LB + AR | 10 | 2013 | Yao[147] |
| *In vivo*, instability with SL, FE | 19 | 2005 | Otani[91] |
| *In vivo*, COR with SL, FE | 33 | 2005 | Schneider[118] |
| *In vivo*, instability with SL, FE | 29 | 2002 | McGregor[75] |
| *In vivo*, overall ROM with SL, FE | 203 | 2001 | McGregor[77] |
| *In vivo*, angular/translational motion + phase lag with SL, FE | 61 | 2001 | Takayanagi[127] |
| *In vivo*, instability with SL, FE | 8 | 2000 | Axelsson[9] |
| *In vivo*, phase lag with SL, FE | 13 | 1998 | Okawa[89] |
| *In vivo*, instability with SL, FE | 50 | 1994 | Wood[144] |
| *In vivo*, instability with SL, FE | 102 | 1991 | Dvorak[24] |
| *In vivo*, instability with SL, FE | 1500 | 1991 | Friberg[27] |
| *In vivo*, angular motion with SL, FE | 6 | 1985 | Pearcy[102] |
| *In vivo*, instability with SL, FE | 202 | 1984 | Saraste[116] |
| *In vivo*, COR with SL, FE | 38 | 1980 | Penning[104] |
| *In vivo*, instability with SL, FE | 4 | 1976 | Olsson[90] |

```
MEDLINE SEARCH:

    Keyword: Motion [Title/Abstract] AND Ligament [Title/Abstract] AND Degeneration [Title/Abstract]
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⇩

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INCLUSION CRITERIA:                 119 Studies

    Time range:    1970-2016
    Method:        In-vivo, all measurement methods
    Data:          Values for Angular/Translation ROM, Overall lumbar ROM associated with LD

                    13 Studies
```

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REFERENCE SEARCH:                          FINAL LITERATURE:
                      4 Studies
                                                     3 Studies
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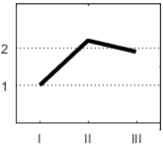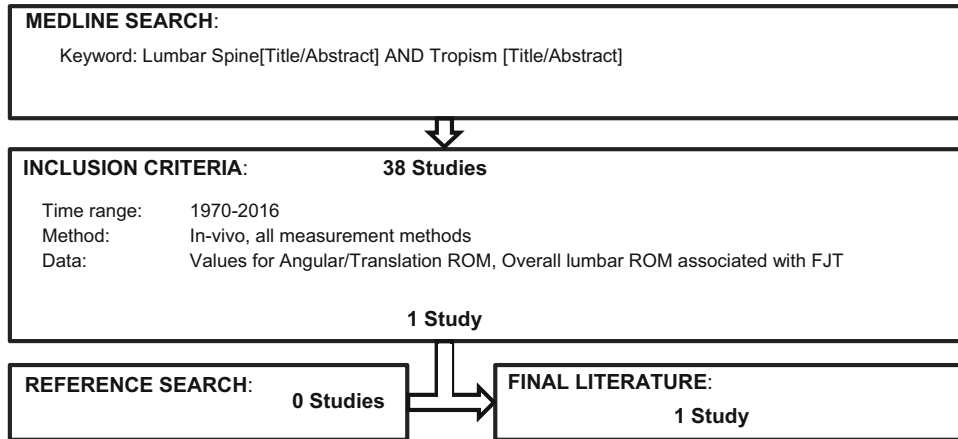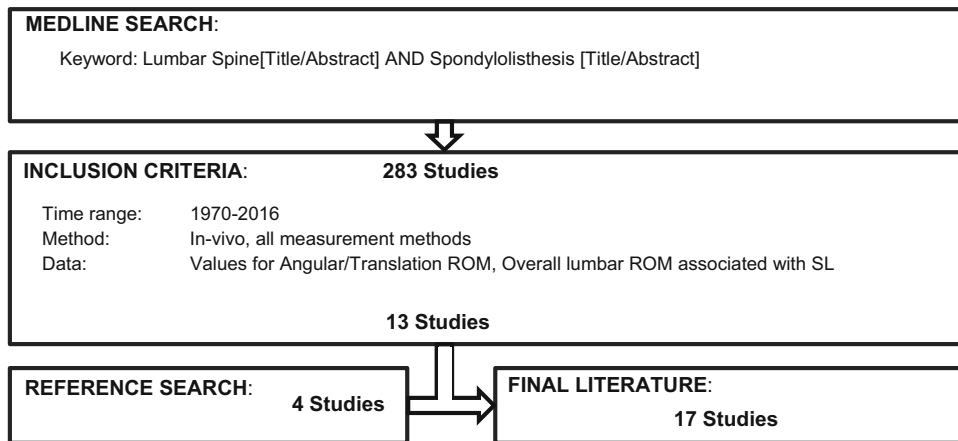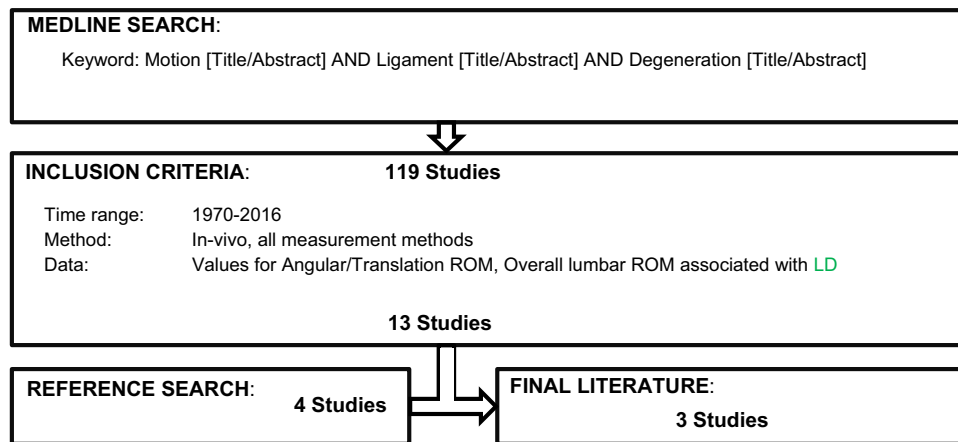
FIGURE 22. Search strategy for studies on spinal ROM and ligament degeneration.

**TABLE 12. Results of the search for literature on spinal ROM and ligament degeneration.**

| Methods | Subject numbers | Years | Authors |
|---|---|---|---|
| *In vivo*, angular/translational motion with ISL degeneration, FE | 64 | 2010 | Keorochana[59] |
| *In vivo*, angular/translational motion with ISL degeneration, FE | 316 | 2009 | Kong[64] |
| *In vivo*, angular/translational motion with LF hypertrophy, FE | 315 | 2009 | Kong[63] |

*Paraspinal Muscle Degeneration*

See Fig. 23 and Table 13.



**MEDLINE SEARCH**:

    Keyword: Lumbar Spine[Title/Abstract] AND (Degenerated[Title/Abstract] OR

    Degeneration[Title/Abstract]OR Degenerative[Title/Abstract]) AND Muscle [Title/Abstract]

**INCLUSION CRITERIA**:     **157 Studies**

Time range:    1970-2016
Method:    In-vivo, all measurement methods
Data:    Values for Angular/Translation ROM, Overall lumbar ROM associated with PMD

**1 Study**

**REFERENCE SEARCH**:     **0 Studies**
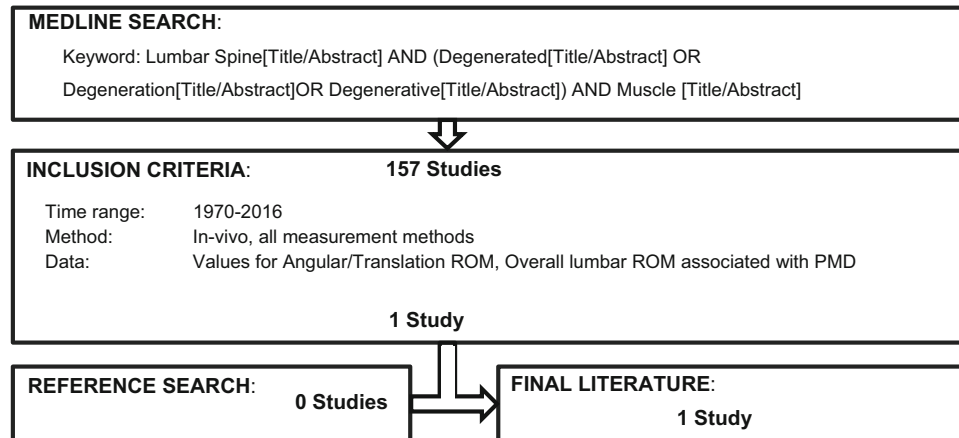
**FINAL LITERATURE**:     **1 Study**

FIGURE 23. Search strategy for studies on spinal ROM and paraspinal muscle degeneration.

TABLE 13. Results of the search for literature on spinal ROM and paraspinal muscle degeneration.

| Method | Subject number | Year | Author |
|---|---|---|---|
| *In vivo*, angular/translational motion with LF hypertrophy, FE | 315 | 2009 | Kong[63] |

## CONFLICT OF INTEREST

No potential conflicts of interest.

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