Etiology of lumbar lordosis and its pathophysiology: a review of the evolution of lumbar lordosis, and the mechanics and biology of lumbar degeneration

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The goal of this review is to discuss the mechanisms of postural degeneration, particularly the loss of lumbar lordosis commonly observed in the elderly in the context of evolution, mechanical, and biological studies of the human spine and to synthesize recent research findings to clinical management of postural malalignment. Lumbar lordosis is unique to the human spine and is necessary to facilitate our upright posture. However, decreased lumbar lordosis and increased thoracic kyphosis are hallmarks of an aging human spinal column. The unique upright posture and lordotic lumbar curvature of the human spine suggest that an understanding of the evolution of the human spinal column, and the unique anatomical features that support lumbar lordosis may provide insight into spine health and degeneration. Considering evolution of the skeleton in isolation from other scientific studies provides a limited picture for clinicians. The evolution and development of human lumbar lordosis highlight the interdependence of pelvic structure and lumbar lordosis. Studies of fossils of human lineage demonstrate a convergence on the degree of lumbar lordosis and the number of lumbar vertebrae in modern *Homo sapiens*. Evolution and spine mechanics research show that lumbar lordosis is dictated by pelvic incidence, spinal musculature, vertebral wedging, and disc health. The evolution, mechanics, and biology research all point to the importance of spinal posture and flexibility in supporting optimal health. However, surgical management of postural deformity has focused on restoring posture at the expense of flexibility. It is possible that the need for complex and costly spinal fixation can be eliminated by developing tools for early identification of patients at risk for postural deformities through patient history (genetics, mechanics, and environmental exposure) and tracking postural changes over time.

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LUMBAR lordosis is unique to the human spine and is necessary to facilitate our upright posture. However, decreased lumbar lordosis and increased thoracic kyphosis are hallmarks of an aging human spinal column.^{75,143} Degeneration of the sagittal spine curvature leads to loss of sagittal alignment, which is implicated in a broad range of adverse health outcomes.⁶⁷ Significant postural degeneration often requires surgical intervention to alleviate pain and facilitate upright posture and ambulation. However, surgical interventions to correct postural deformities are costly and complicated and include a risk of neurological deficits. The unique upright posture and

lordotic lumbar curvature of the human spine suggest that an understanding of the evolution of the human spinal column and the unique anatomical features that support lumbar lordosis may provide insight into spine health and degeneration.

Evolutionary medicine is being touted as a more holistic way to understand and treat conditions of the human body.^{55,206} While this approach is primarily targeted toward understanding drug therapies and genetics, the approach is also valuable for understanding age- and disease-related degenerative changes to the body structure. Considering evolution of the skeleton in isolation from other scientific studies provides a limited picture for clinicians. Conducting a review of the evolution of lumbar lordosis within the context of recent findings in spine mechanics and the biology of spinal structures will help to

Abbreviations used in this paper: LL = lumbar lordosis; mya = million years ago; PI = pelvic incidence; ROM = range of motion; SVA = sagittal vertical axis.

influence our clinical management of lumbar pathophysiology and suggest effective pathways for intervention.

The goal of this review is to discuss the mechanisms of postural degeneration, particularly the loss of lumbar lordosis commonly observed in the elderly in the context of evolution, mechanical, and biological studies of the human spine and synthesize recent research findings to our clinical management of postural malalignment. Degenerative conditions in the lumbar spine have been attributed to the overuse,¹⁸⁶ underuse,¹⁸⁴ and misuse¹²³ of the spine as well as being identified as an evolutionary “mismatch” (between our evolved selves and our current lifestyle).^{60,113,116} Understanding the evolution of lumbar lordosis and the anatomical features that facilitate lordosis in hominins, both extant and extinct, may assist in determining optimal uses of the spine and identify opportunities to reduce degenerative changes. Reviewing these evolutionary theories in the context of spine mechanics and biology will provide unique insights into postural degeneration that should highlight opportunities for clinical intervention.

Evolution of Lumbar Lordosis With Habitual Bipedalism

Extensive research has examined the origins of our habitual bipedalism by looking into the fossil record and noting significant morphological changes necessary to support upright posture. A key challenge in mapping the progress of evolution is the limited number of specimens and the poor condition of most samples. Unlike medical research where statistical significance is critical for conclusions, the foundation of evolution research is the development of theories or hypotheses based on scant physical artifacts. We provide a very brief summary of lumbar spines within the fossil record and the morphological trends in lumbar spine curvature that begins from a presumably generalized nonlordotic lumbar spine of a last common ancestor with chimpanzees to the fully lordotic lumbar spines of modern humans.

The divergence between extant apes occurred 5–11 million years ago (mya) between *Homo* and *Pan* (includes chimpanzees and bonobos).¹⁷⁷ Although morphologically and genetically similar to humans, extant great apes are orthograde quadrupeds that demonstrate a unique “knuckle walking” locomotion and are capable of limited durations of bipedality. Despite many structural similarities in the spinal columns and vertebrae, the great apes’ lumbar spines lack a lordotic curve typical of humans. The flat lumbar spines of the great apes are also much stiffer than human spines and offer limited mobility.¹¹⁶ Understanding the functional limitations of a flattened lumbar spine, typical in the great apes, may provide insight into the effects of degeneration lumbar flattening in elderly humans.

Habitual bipedalism is currently shown to date back to *Ardipithecus ramidus* (4.4 mya) with morphological features indicative of bipedalism, such as an acute greater sciatic notch and a facultative medial longitudinal arch of the foot.¹¹⁸ Older potential hominin bipeds include

Orrorin tugenensis from Tugen Hills, Kenya, at roughly 5.8–6.1 mya¹⁷⁵ and *Sahelanthropus tchadensis* from South Sahel, Chad, at 6–7 mya;³⁹ however, fossil evidence for lumbar morphology is scarce due to the porous and largely trabecular composition of lumbar vertebral bodies. The earliest lumbar spines include partial spines from *Australopithecus africanus* (Sts 14; Stw 431)^{157,189} at 2.5–3 mya, *Australopithecus sediba* (MH1 and MH2) at 1.977 mya,²⁰⁷ and *Homo erectus* (KNM-WT 15000, “Turkana boy”) at 1.5 mya.³⁸ There is debate about the number of lumbar vertebrae in australopithecines. Some analysis of the partial spines suggest 6 lumbar vertebrae,^{117,157} while other reviews indicate 5 lumbar vertebrae, when counting from the last rib-bearing vertebra, similar to modern humans.^{73,74,207} The one *H. erectus* juvenile is also thought to have 5 lumbar vertebrae.^{73,74} All of these early partial lumbar spines demonstrate a variably mild degree of lordosis, based on the amount of wedging on the posterior border of the lumbar vertebral bodies.^{24,107,157,200,204,207}

Estimates of the degrees of lumbar lordosis in hominin ancestors have been difficult to obtain because of the absence of the intervertebral discs and the lack of complete spine specimens. A new method for calculating the lordotic angle based on the orientation of the inferior articular process explained 89% of the variation in lordotic curvature in humans and primates.²¹ Applying this method to the spines of extinct hominins showed the lordotic angles of australopithecines ($41^\circ \pm 4^\circ$), *H. erectus* (45°), and fossil *Homo sapiens* ($54^\circ \pm 14^\circ$) were similar to modern humans ($51^\circ \pm 11^\circ$), while modern nonhuman apes had smaller lordotic angles ($22^\circ \pm 3^\circ$) than humans.²⁴ Interestingly, despite being contemporary with *H. sapiens*, *Homo neanderthalensis* showed significantly smaller lordotic angles ($29^\circ \pm 4^\circ$) than humans, which may suggest differences in Neandertal posture and locomotion from that of modern humans.²⁴

Considering the close genetic relationship and anatomical similarities between Neandertals and humans, it is quite surprising that the lumbar spine of Neandertals is hypolordotic. The lumbar lordosis of early genus *Homo* falls between the lesser lordosis of Neandertal and greater lordosis of humans.²⁴ The reduced form of lordosis in the Neandertal spine is attributed to an anterior wedging of the lumbar vertebral bodies.^{21,26,202} The functional reason for the reduced lordosis of the Neandertal lumbar spine is unclear; however, it may provide a mechanical benefit for locomotion in sloped terrain. Neandertals are thought to have spent significant time on sloped, mountainous terrain in Eurasia,⁸⁴ and gait studies on modern humans demonstrate a significant lumbar flattening during uphill walking.^{110,111,197}

Structural Evolution in the Lumbar Spine

The distinct lordotic curve of the human lumbar spine is created by wedging of both the lumbar intervertebral discs and the vertebral bodies. A study by Been et al.²² measured the relative contributions of vertebral and disc wedging on lordotic curves in humans and pronograde primates (macaques). On average, approximately 10% of the lumbar curve is contributed by wedging of

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the vertebral bodies (5°), while the remaining 90% (46°) is due to wedging of the discs. In contrast, macaque vertebral body wedging in the lumbar spine opposes lumbar lordosis. The average lordotic curve in a macaque is 15° while the wedging of the vertebral bodies contributed 25° of kyphosis. The lumbar discs in the macaques, however, showed similar lordotic wedging as seen in the human spines (40° vs 46°). These results suggest that the evolution from pronograde to orthograde posture resulted mainly from an increase in the vertebral body wedging and emphasize the important contribution of discs in maintaining lumbar lordosis. The evolution of lumbar lordosis to the degree at which it is seen in modern humans occurred in parallel with other skeletal changes such as limb lengthening,^{10,154,155} pelvic restructuring,^{116,162} thoracic invagination of the spine,¹¹⁶ and reorganization of the spinal musculature to support bipedal motion.

Changes to the curvature of the lumbar spine also required adaptations within the spinal musculature, including migration of key muscle attachments on the vertebral body and the spinopelvic structure. A comprehensive review of the fossil records revealed two important structural changes to the lumbar vertebrae.⁶⁰ The first change was the posterior shift of the lumbar transverse processes from the vertebral body, as they continue to be in Old World Monkeys, to the neural arch, though the timing of migration is debated.¹⁶⁰ The shift of the transverse processes allowed the longissimus muscles to become major lateral flexor and extension muscles of the spine, necessary for rotating the pelvis and maintaining balance during bipedal walking,⁶⁰ and limited the role of the erector spinae muscles to resisting forward flexion.¹⁶⁶ The second change was the loss of the styloid processes. The loss of the styloid processes facilitated a larger range of motion (ROM) in the hominin spine, allowing for adoption of a variety of postures. Later changes to the spinopelvic structure included a widening of the sacrum and ilia and a progressive cranial to caudal widening of the lumbar vertebrae.¹¹⁶ The structural changes to the vertebrae preceded changes in musculature in the spine. The migration of the insertion points of the iliocostalis lumborum muscles to the iliac crests, necessary for spine stabilization and rotation, occurred 15–18 mya.⁶⁰ The posterior migration of the posterior superior iliac spine (1–2 mya) combined with the iliocostalis lumborum migration facilitate modern human lumbar lordosis.⁶⁰

The connection between the sacrum and the pelvis provides the anchor point for the lumbar spine and a means to translate the load of the upper body to the pelvis and lower limbs.¹⁴⁷ Simultaneous to changes in the vertebrae and back musculature during evolution, there were significant changes in the pelvis. These changes in the pelvis supported upright posture and efficient bipedal motion as well as increasing the diameter of the birth canal. A recent study of human, hominin, and hominoid pelvises was the first to characterize changes in pelvic incidence throughout evolution.²⁸ Pelvic incidence (PI) is a fixed clinical measure of sacral orientation in the pelvis (Fig. 1). Hominoids in the study included *Pan*, *Gorilla*, *Pongo*, and *Hylobates*. The PI for hominoids was consistent for all species ($27^\circ \pm 5^\circ$). This was substantially

Fig. 1. Illustration showing the standard angles of the spine and pelvis. Pelvic incidence is the angle between a line connecting the center of the sacral plate to the middle axis of the femoral heads and a line perpendicular to the midpoint of the sacral plate. Sacral slope (SS) is the angle between the sacral plate and a horizontal line. Pelvic tilt (PT) is the angle between the line that connects the midpoint of the sacral plate to the middle axis of the femoral heads and a vertical line. Summing PT and SS equals the PI. The PT and SS are oppositely affected by changes in patient stance. However, PI is a fixed measure, independent of patient stance or posture. Copyright Andrew McNichol. Published with permission.

lower than the PI measured in australopithecines ($43.5^\circ \pm 2^\circ$) and modern humans ($54^\circ \pm 10^\circ$). Interestingly, the PI for Neandertals was similar to hominoids, not modern humans. There was a strong correlation between PI and lumbar lordosis for each group of hominoids, hominins, and modern humans (Table 1). The pelvic structure of australopithecines and Neandertals are wider than current modern humans. The increased sacral angle in modern humans was necessary to enlarge the pelvic outlet to accommodate large fetal heads.²⁰⁰ The close correlation between PI and lumbar lordosis is a mechanism for the body to position its center of gravity. However, PI may be compromised between the evolutionary pressures for efficient bipedal motion and the obstetric requirements of modern humans.

TABLE 1: Pelvic incidence and lumbar lordosis (L1–S1 Cobb angle) in normal adults correlate with each other in hominoids, hominins, and modern humans*

Authors & Year	Species	No. of Samples	PI (Mean $\pm$ SD)	LL (Mean $\pm$ SD)
Been et al., 2013	hominoids	19	27 $\pm$ 5	22 $\pm$ 3.4
Been et al., 2013	<i>Australopithecines</i>	2	43.5 $\pm$ 2	41 $\pm$ 4
Been et al., 2013	Neandertal	3	34	29 $\pm$ 4
Been et al., 2013	modern <i>H. sapiens</i>	53	54 $\pm$ 10	51 $\pm$ 11
Labelle et al., 2004	modern <i>H. sapiens</i>	160	52 $\pm$ 5	NA
Vialle et al., 2005	modern <i>H. sapiens</i>	300	56 $\pm$ 10	58.5 $\pm$ 10
Peleg et al., 2007	modern <i>H. sapiens</i>	255	55 $\pm$ 13	NA
Hong et al., 2010	modern <i>H. sapiens</i>	30	NA	39.88 $\pm$ 10.02

* There is large variation in normal measures of PI and lordosis, which are measured in degrees. LL = lumbar lordosis; NA = not available.

Degeneration of Lordosis: Misuse, Overuse, or Evolutionary Weak Point?

The human lumbar spine is often labeled the “evolutionary weak point” of the spine and is the most common site of degenerative changes in the vertebrae and intervertebral discs.⁶⁰ Particular focus is given to the human L5–S1 junction as the spinal segment with the greatest individual curvature and the greatest occurrence of degenerative conditions (20% of the total spine).⁶⁰ Lumbar lordosis is unique to the human spine, which fuels this accusation of evolutionary failure. However, degenerative changes occur with similar frequency in spines of primates in captive populations despite their lack of lumbar lordosis.¹⁴⁰ In contrast, wild populations of old world primates show a remarkable absence of degeneration.⁹⁵ The challenge of accurately aging wild primates makes it difficult to determine if degenerative spinal conditions in captive populations are a result of longer life spans in captivity or forced changes in lifestyle. Interestingly, captive populations of macaques are reported to spend a significant portion of their waking time sitting, unlike their wild counterparts.¹⁴⁰

The occurrence of degenerative conditions in human spines motivates the question of whether humans are now outliving their evolved form. Over 3 million years ago the maximal lifespan of our human ancestors was similar to that of great apes, approximately 50 years.⁷⁹ In reality, the average life expectancy was much lower due to high rates of infant and child mortality. The average human lifespan increased dramatically by reducing infant/child mortality and reducing disease and war-related mortality in adults in the Upper Paleolithic era (30,000 years ago).⁴² Increased longevity after the cessation of reproduction means that degenerative spine conditions do not factor into direct selection. However, there are theories that support the evolutionary benefits of postreproduction longevity in women. These theories highlight the survival advantages of her children¹⁴⁶ and grandchildren.^{78,79,142} In addition, evolution models project the current maximal human life span at approximately 100–120 years.⁹⁷ It therefore seems unlikely that spinal degeneration occurring in patients as young as 30 years is a result of people outliving their evolved design.

The human lumbar spine has adapted its configuration throughout evolution. Skeletons of *Australopithecus* showed lumbar spines that likely comprised 6 lumbar vertebrae.^{117,128,203,207} In the subsequent course of evolution the human lumbar spine was reduced by 1 vertebra when that vertebra was captured in the pelvic girdle and became part of the sacrum. In a review of the Galloway Osteological Collection (Kampala, Uganda), 4% of modern humans continue to have a sixth lumbar vertebra.¹²⁰ A classification that looks at the spine as a continuum in humans and extant primates indicates the total number of vertebrae in the thoracolumbosacral spine is consistent, with 81% of samples having 22 vertebrae, while 19% of samples had 21 or 23 vertebrae; it is the distribution of vertebrae among the thoracic, lumbar, and sacral regions that is species specific.² In gorillas the lumbar spine evolved to 3 (41%) or 4 (38%) vertebrae, while the thoracic spine has 13–14 vertebrae.² This truncated lumbar spine coupled with long ilia of the pelvis supports the substantial bulk of the gorilla’s upper body and effectively shields the lumbar spine from overloading.¹²⁸ The short lumbar spines of great apes are very stiff and have limited flexion.²⁰¹ This differential evolution of human and old world primate lumbar spines suggests a functional purpose for 5 lumbar vertebrae in humans, not an evolutionary failure.

Determining the effect of modern lifestyle on spinal degeneration in humans is difficult. However, several studies examining osteological remains around the globe show a common pattern of osteoarthritis and osteophytosis in the cervical and lumbar spine in specimens dating from 3500 years ago³⁵ to postmedieval times 800 years ago,¹⁵⁸ suggesting that spinal degeneration cannot be solely attributed to the sedentary lifestyle of the average westernized society. In contrast, in Indian tribes with minimal Western influence, elderly tribesmen demonstrated little to no disc degeneration in their spines.⁵⁹ Despite limited disc degeneration, however, there was substantial degeneration in the vertebral bodies similar to the previously cited studies. The study noted that the tribesman rarely stood in a static posture. If not walking the tribesmen would squat (not sit), which is a dynamic posture requiring continuous stabilization. The elderly tribesmen also

demonstrated slightly less lumbar lordosis than their European counterparts. These observations of spine and disc health within isolated populations motivate discussion of another element of disc and spinal degeneration. Are differences in disc health and longevity observed dictated by lifestyle (mechanical loading) or genetic selection in these different populations? In the subsequent sections we review the recent literature on lumbar spine mechanics and vertebrae and disc biology to further understand degenerative lumbar flattening.

Sexual Dimorphism and the Lumbar Spine

Accounting for sexual dimorphism is becoming increasingly important and common in many fields of medicine and must also be considered in this discussion of lumbar lordosis. Upright, bipedal posture brings a unique set of challenges to the female spine during pregnancy. The increased fetal load anterior to the spine changes the center of mass of the trunk and could affect ambulation and balance if there was no adaptation of the pregnant mother's stature. A recent study demonstrated a significant increase in lumbar lordosis during pregnancy and a change in load distribution in the lumbar spine to have the zygapophysial joints carry more than double their normal load during pregnancy.²⁰⁴ This shift in load distribution is thought to shield the discs from damaging shear loading. Three key structural differences between male and female lumbar spines facilitate this lordotic adaptation. The first is that lordosis attributable to vertebral body wedging is greater in females than in males.^{126,204} Second, female zygapophysial joints are larger relative to vertebral body size (14%) and more coronally oriented (13%) than males. Third, the relative interfacet distance was wider in females than males.¹²⁶ Several other studies have observed the degree of standing lumbar lordosis in the female spine to be 26%–28% greater than the male spine;^{30,139,210} however, no information was provided on the parous history of those females. Interestingly, changes in mass distribution in the body due to increased body mass index showed little^{85,137} or no effect on lumbar lordosis in males or females.^{159,211}

Development of Lumbar Lordosis

The physical capacity of the human spine to develop lumbar lordosis is due to evolutionary changes in the spine and pelvis; however, the degree of lumbar lordosis develops through infancy, toddler, and childhood. Human infants show little or no lumbar lordosis in utero,⁴⁴ and the degree of lumbar lordosis coincides with the stages of bipedal activity in modern humans.¹ Other researchers have demonstrated that the degree of lumbar lordosis continues to increase through childhood.⁴⁶ Lumbar lordosis alone is not sufficient to support the ROMs the human body undergoes on a daily basis; the spine must also be flexible. Lumbar lordosis can be developed (to a lesser extent) in primates trained to walk bipedally from infancy;^{80,151} however, the lumbar curve in these primates is fixed and does not have the flexibility of the human spine.

Mechanics of Lumbar Lordosis

Mechanical Advantage of Lumbar Lordosis

To understand the potential underlying causes of degeneration it is important to know the functional benefits of lumbar lordosis. Lumbar lordosis is critical for balancing the human body in upright posture. However, lumbar lordosis is not a uniquely human trait. In infants the lumbar spine has only slight lordosis or may have no lordosis at all. The lordotic curve develops with developmental stages of bipedalism.¹ Infants and toddlers who walk early demonstrate increased lordotic curves while those who walk late or not at all have only slight lordosis.¹ Japanese macaque monkeys trained from infancy to walk bipedally also demonstrate lumbar lordosis.¹⁵¹ The critical distinction between human and primate lumbar spines, however, is the flexibility or ROM in the spine. Macaques' lordosis develops from an increase in the ventral lengths of the vertebral bodies without corresponding variations in spinous process alignment; therefore, they maintain their lumbar lordosis during all activities including sitting. In contrast, humans show a significant flattening of their lumbar spines during relaxed sitting.¹⁵¹ While lumbar lordosis is necessary for efficient upright walking, lumbar flattening is equally necessary for other activities. Understanding the influence of lumbar lordosis and lumbar flexibility on human activities is critical for evaluating how to best balance the needs of a patient when determining the most effective approach to correct the curvature of a deformed spine.

The degree of lordosis in the lumbar spine is the main factor that influences the conversion of the extensor power developed by the intrinsic back muscles to axial torsion necessary to rotate the pelvis in walking. Primates without lumbar lordosis who occasionally engage in bipedal walking demonstrate great lateral pelvic movements because of the inability to rotate their pelvises.⁹¹ However, a chimpanzee with forelimb paralysis who adopted a bipedal gait in the wild did show an adaptation to rotate the pelvis during gait.²⁰ This change occurred along with a significant decrease in thoracic kyphosis and retraction of the scapula, which moved the center of mass over the pelvis. The rotation of the pelvis during normal human gait comes primarily from flexibility in the lumbar spine. In healthy people, the pelvis rotates an average of 10.4° during walking, with 8.34° attributable to lumbar axial rotation.²⁰⁵ Lumbar spine flexibility or ROM provides mechanical advantages for sitting, lifting, and bending tasks by changing the distribution of loading in the spine.⁴ In healthy spines the lumbar spine flattens 40°–43° during sitting^{51,167} and flexes an average of 40° during lifting.¹²² Although body mass index does not correlate with the degree of lumbar lordosis, it is strongly correlated with lumbar spine ROM.^{32,65,125,134,208} High body mass index limits lumbar ROM in pediatric patients²⁰⁸ and in workers lifting objects⁶⁵ and is particularly limiting in seated postures.¹³⁴

There are significant mechanical implications of degenerative lordosis. Mechanically, postural degeneration becomes a positive feedback cycle of worsening conditions. In a healthy spine the center of rotation at each spinal level is within the vertebral body during passive

standing (Halverson P, Bowden A, Stratton E, et al., presentation at the 55th Orthopaedic Research Society Annual Meeting, February 22–25, 2009, Las Vegas, NV), meaning muscles are only acting to stabilize the spine but not support the mass of the head, arms, and trunk. Relaxed standing posture, when balanced, is only 7% more energetically expensive than supine relaxing.¹²⁷ However, slight changes in sagittal balance resulting from postural changes lead to increased muscle activity in the erector spinae to oppose the forward rotation of the body.⁵⁷ The increased muscle activity increases the compression in the spine, leading to creep in the intervertebral discs. The loss of disc height from constant loading brings the facet joints closer together. With time, the facet joints become close enough to touch and begin carrying a portion of the spinal load.⁵² Altering the load sharing in the lumbar spine due to disc degeneration shields the anterior vertebral body from loading in upright postures and reduces the bone volume fraction in the vertebral body.⁶ However, in flexed postures loading in the anterior vertebral body is independent of disc morphology. This can lead to overloading in the weakened anterior vertebral body in flexion and spine fractures.

Mechanics in the lumbar spine are of particular interest because lumbar vertebrae and intervertebral discs experience the greatest mechanical loads in the spine. They are also the most common site of degenerative changes and low-back pain.¹⁹ Mechanical loading of the lumbar spine has been studied extensively in isolated in vitro preparations. These studies have shown that the distribution of loading across the vertebrae is a function of posture¹⁰⁸ and spine health.^{6,150} In a healthy spine the neural arch carries negligible compressive load.⁵² However, in a degenerative spine subjected to prolonged compressive creep loading in a standing posture, the neural arch may resist up to 70% of the compressive force.⁵ The effects of posture on load distribution in the spine become exaggerated when disc height is reduced by pathological changes or sustained loading.⁵²

Musculature and Lumbar Lordosis

The degree of lumbar lordosis may be constrained by vertebral body wedging and disc morphology; however, spinal muscle strength also dictates the degree of lordotic curve.¹⁷⁸ At the extreme, activities that result in extensor muscle atrophy such as farm work in hyperflexed postures for long periods (9–10 hrs/day) or extended bed rest result in a significant loss of lumbar lordosis.^{29,186} Conversely, overuse and overdevelopment of musculature has also been shown to correlate with disc degeneration.¹⁴ In patients with chronic low-back pain, targeted back exercises proved less effective than walking for reducing pain.⁸⁷ Walking introduces dynamic motion in the spine and instability that requires moderate spine extensor muscle activity (4%–13% of their maximum).¹³⁵ In contrast, simple isometric back exercises result in much greater extensor muscle activation (24%–42% of maximum).⁷¹ Sitting results in reduced muscle activation that correlates with lumbar spine flexion.¹³⁶ Slouched, seated postures resulted in extensor muscles shutting off almost completely, a phenomenon called flexion relaxation.^{41,48,136} It is likely that preserved spinal

posture is achieved through finding an optimum balance of strength and flexibility of the joints.

Spine biomechanics research indicates that the extensor muscles generate a follower load. This refers to the resultant force that is tangential to the sagittal spinal curvature and allows the lumbar spine to support the weight of the upper body. The extensor muscles are also critical for maintaining stability during movement.^{11,76,101,145} Recent modeling suggests that a spine with large lordosis requires a greater follower load in the standing position than one with minimal lordosis.¹³⁰ Since muscle force is proportional to muscle volume,^{7,33,61,94} increased lumbar lordosis requires larger extensor musculature to provide sufficient follower loads and sagittal stability. Previous studies showed the degree of lumbar lordosis correlated with extensor muscle volume¹³¹ and extensor muscle strength,¹⁷⁸ while decreased extensor muscle volume correlated with back pain.^{50,83,96,131,198} There is strong evidence that early intervention may be effective in correcting posture problems in young subjects by improving core muscle balance.¹⁶⁷ However, the long-term outcomes of such an intervention are unknown. In addition, it is unclear if muscle strengthening could be equally effective in older patients or those showing early signs of postural degeneration.

Whether the relationship between lumbar lordosis and extensor muscle size is causative or correlative is not yet clear. Several studies have shown that spine shape correlates with vertebrae morphology and orientation of the posterior elements.^{27,132} Variations in these parameters affect the forces required to stabilize the spine, which may manifest as changes in extensor muscle volume. Conversely, variations in muscle size may be the driving force in lumbar curvature. Several studies have shown a reduction in lumbar lordosis believed to be associated with age-related loss of muscle mass;^{75,185} however, this may also be explained by other age-related changes such as decreased disc height, anterior wedging, or increased spinous process height.^{12,185} The relationship between lumbar lordosis and spinal musculature is complex and warrants further anatomical and clinical analysis.

Perturbing the Balanced Spine

Maintaining upright posture requires that the center of pressure due to the mass of the standing person be contained within his or her base of support (Fig. 2). Human subjects have shown a variety of adaptive strategies to ensure balanced standing in the face of changing postures. Experiments on healthy subjects walking and standing in a variety of postures suggest that increased muscle activity and energy expenditure result from stooped posture. In one study, healthy subjects were asked to maintain upright, 25° and 50° flexed postures during standing while their energy consumption was monitored.¹⁶⁴ To maintain balance and keep their center of pressure in the same fore-aft position under their base of support, the subjects employed a number of compensatory strategies including increased hip flexion and ankle plantarflexion and increased cervical spine lordosis, which resulted in increased oxygen consumption (25° and 50° flexion increased consumption by 28% and 60%, respectively, over

Fig. 2. Cone of economy and center of pressure (CoP). The cone of economy concept is based upon a cone centered at the foot of a standing patient projecting upward and outward. Alignment within the center of this cone is ergonomically favorable while excessive deviation in the anteroposterior or lateral planes requires increased energy expenditure to maintain balance and may produce fatigue, pain, or disability (Dubouset⁵⁴). The center of pressure demonstrates the same mechanics in a different manner. If the center of pressure (X) is centered within the base of support (shaded area), the person is most stable; as the center of pressure moves toward the edges of the base of support, a person is less stable and it is energetically more costly to maintain upright posture.

upright standing). Standing subjects who were directed to change their pelvic tilt angle showed an unconscious equivalent but opposite change in spinal curvature to compensate for the change in balance.¹¹² Subjects asked to walk with flexed postures employed a bent knee, bent hip strategy similar to primates.¹⁶³ This bent knee, bent hip strategy resulted in a 10% shorter stride length and an increase in steps per minute to maintain the same walking pace as the upright posture. These findings are seconded by experiments in chimpanzees trained to walk bipedally.¹⁸³ For the majority of chimpanzees, bipedal ambulation with stooped posture results in bent knee, bent hip gait; however, in a subset of chimpanzees a different biomechanical approach was used that resulted in bipedal motion being more energy efficient than quadrupedal walking (though the bipedal chimps were still significantly less efficient than bipedal humans). Understanding that even slight changes in posture or the biomechanical approach to walking with compromised posture may affect bipedal efficiency is an important indicator of the need for early intervention in postural degeneration. Prolonged bent knee, bent hip gait, or excessive energy expenditure is likely to exacerbate postural degeneration in addition to triggering degeneration in other joints.

Postural correction and balance within the spine appears to be an unconscious response moderated by neurocontrols. In patients with postural deformities, the strategies to compensate for a loss of sagittal balance are consistent across individuals. Those with moderate lumbar flattening compensate by further extending their thoracic spine and hyperextending their hips.⁷⁷ Patients with lumbar degenerative kyphosis who employed these compensatory strategies preoperatively showed an immediate resumption of normal thoracic kyphosis (increased 16° postoperative) and sacral slope (increased 14° postoperative) despite only their lumbar curvature being surgically restored.⁹³ Deficits in proprioception and neurocontrol have been correlated with scoliotic deformities in growing children.¹¹⁹ However, little is known about the relationship between neurocontrol and postural degeneration in older patients.

Healthy individuals also employ unconscious postural correction strategies to maintain balance. During running, the pelvic tilt increases 15° to 20° to facilitate an increased stride length.¹¹² The torso angle changes opposite to the pelvic angle to maintain the center of pressure within the base of support. In both walking and running the lumbar spine oscillates with each step. The magnitude of oscillation is 4°–9° for walking¹¹¹ and 5°–21° for running.^{111,168} Maximum flexion of the lumbar spine correlated with toe off in the gait cycle while maximum extension occurred near the timing of heel strike.¹⁶⁹ Walking and running both result in cyclic impacts to the body with each step which in turn applies a cyclic (or dynamic) load to the spine.

Mechanobiology

Researchers now widely recognize links between mechanical loading and the biological responses of human tissue. Long before structural failures (for example, fracture or disc herniation), significant biological changes are occurring in tissues as a result of mechanical loading. Mechanobiology may be either positive or negative. Load-bearing structures in the body, such as bones, cartilage, and discs, depend on mechanical loading to trigger biological processes that keep them healthy; however, excessive or abnormal mechanical loads may lead to detrimental adaptations in the tissues of the lumbar spine.

Several studies have characterized the effect of spinal degeneration on load distribution in the tissues (for review see Niosi and Oxland¹³⁸). It is hypothesized that osteoporotic fractures in the spine may be a long-term result of changing load distributions in the spine. Slight variations in spinal curvature (2°) drastically reduce the loads seen by the vertebral body and disc and result in greater loads carried in the vertebral arches.⁵ Reduced loading in bone is known to lead to bone resorption.⁶³ In the spine this may lead to resorption of bone in the vertebral bodies. In flexed postures, the vertebral arches are not loaded and the majority of compressive loading is carried by the weakened vertebral bodies, which may lead to osteoporotic fractures in the vertebrae.^{6,150}

Seated posture requires flexion in the lumbar spine and concentrates the compressive load in the vertebral

bodies and intervertebral discs. However, there is little evidence in the spine biomechanics literature that this concentration of load in the disc during sitting is sufficient to cause injury.⁴⁷ Static disc pressures, such as those occurring in prolonged standing or sitting, are, however, likely to disrupt the flow of nutrients into the disc,^{176,199} which may have implications for long-term disc health.

The intervertebral disc is an avascular structure; therefore, the flow of nutrients into the disc and removal of waste products from the disc requires convective transport and diffusion. While diffusion is the dominant factor in nutrient delivery,¹⁹¹ fluid flow out of the disc during normal daily activities appears to be a primary factor in waste removal.¹²⁴ Normal daily activities result in a combined disc height loss of 13–21 mm^{144,156} and an average fluid loss of 0.9–1.3 cm³ per disc per day^{124,156} in healthy spines. Walking with a heavy load (40% of body weight) produced an equivalent height and fluid loss in only 4 hours.⁸⁸ Comparing the effects of different activities on fluid loss in the disc showed sitting resulted in 40% less disc height loss than walking.¹⁹³ Although excessive disc height loss may be detrimental to disc health, height loss is necessary to generate pressure gradients that expel fluid (and remove waste) then allow for fluid influx during a diurnal cycle.

In addition to the magnitude of loading, the biological response of the intervertebral discs to mechanical loading is highly rate dependent. Mechanical studies demonstrate that dynamic loading on the disc at the frequency and loading magnitudes typical of bipedal walking or running increase nutrient flow.¹¹⁴ In contrast, static, high rate, or high magnitude loading are all detrimental to disc nutrition.^{184,199} Dynamic mechanical loading on a hydrated material such as the disc results in increased material stiffness when compared with static loading.⁹⁸ Increased material stiffness leads to less deformation in the disc for a given load, protecting the disc from excessive height loss during dynamic activities such as walking.

Biology

The most common causes of degenerative lumbar deformity are osteoporosis and degenerative disc disease.⁴⁹ Although historically viewed as a result of mechanical overuse, the Twin Spine Study demonstrated a significant role for genetic factors and a high heritability of lumbar disc degeneration, while environmental risk factors such as smoking and increased physical loading had surprisingly modest effects.^{17,18,165} Genetics explained 61% of the variation in disc degeneration in the upper lumbar spine (T12–L4) and only 32% in the lower lumbar spine (L4–S1). Aging and mechanical exposure combined explained 16% of the variation in the upper lumbar spine and 11% of the variation in the lower lumbar spine. In the lower lumbar spine, 57% of the variation in disc degeneration is not explained by genetics, mechanics, or age.¹⁹ A limitation of the Twin Spine Study is that it studied adult twins; therefore, variation attributed to genetics represents the upper limit of genetic influence and incorporates genetics and environmental exposures through childhood.¹⁹ Genetic studies led to the identification of two polymorphisms in the *VDR* gene associated with disc degeneration.¹⁹⁵ Recent

work also identified associations with genes encoding collagen, interleukins, and matrix metalloproteinases.¹⁹ Interestingly, the same research group who determined genetics to be the dominant factor in disc health found positive effects of moderate routine loading on disc health.¹⁹⁶ This research coupled with earlier evolution research that observed marked difference in disc disease in tribesman compared with European males suggests that differences observed in these early populations may have been an effect of controlled genetic pools and not lifestyle.

Osteoporosis has a complex etiology that is determined by genetics and is modulated by hormonal, environmental, and nutritional factors.⁶⁴ Vertebral wedge fractures are a common result of osteoporosis and contribute to overall postural degeneration. Up to 90% of spine and hip fractures in elderly women and 70% in elderly men may be attributed to osteoporosis.¹³³ Osteoporosis is defined as bone mineral density values that are more than 2.5 standard deviations below the young adult average.²⁰⁹ Bone mineral density is the major determinant of bone strength and osteoporotic fracture risk.²⁰⁹ Genetics account for 50%–80% of the variation in individual bone mass.^{149,179} Genetic studies have identified *vitamin D receptor (VDR)*,¹⁸⁸ *collagen Type I $\alpha 1$ (COC1A1)*,⁷⁰ *estrogen receptors (ESR1)*,⁸² *transforming growth factor $\beta 1$ (TGFB1)*,¹⁰⁵ *lipoprotein receptor-related protein 5 (LRP5)*,⁶⁹ *sclerostin (SOST)*,¹⁹⁰ *TCIRG1*,¹⁸² and *CLCN7*¹³ to be associated with osteoporosis. There are multiple polymorphisms within each gene that show varying degrees of disease linkage with osteoporosis.^{64,153} Studies in the field of genetics and osteoporosis have mostly been underpowered, leading to conflicting results and findings that cannot be replicated. Large-scale, longitudinal studies combined with computation bioinformatics will assist in identifying and validating the effects of gene polymorphisms in bone mineral density regulation and osteoporosis risk. Integrating genetics into population screening for osteoporosis will only be warranted once specific genetic links have been clearly established.

Genetics are also linked with an individual's degree of lumbar lordosis and ROM. A recent study of monozygotic twins, dizygotic twins, same-sex siblings, and different-sex siblings showed a strong correlation between the degree of lumbar lordosis and genetic similarity.⁵³ Monozygotic twins showed a high correlation in the degree of lordosis ($R = 0.65$), while opposite-sex siblings showed low correlation ($R = 0.14$). In another study, ROM for normal lumbar spines was also shown to vary by race, with Turkish men showing greater than 25% decrease in lumbar flexion⁴⁵ compared with British men.¹

Clinical Discussion

Given the importance of lumbar lordosis in the evolution of the human spine and its role in our transition to bipedalism, it is not surprising that loss of sagittal balance contributes to significant pain and disability.^{67,104,170} Sagittal malalignment also results in increased energy expenditure and induces a variety of compensatory measures including knee flexion, pelvic retroversion, and thoracic hypokyphosis.^{104,171,172} Studies have shown that

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surgical correction of sagittal malalignment leads to improvements in a variety of health-related quality of life measurements.^{67,172} In patients with lumbar degenerative kyphosis, surgical restoration of lumbar lordosis results in spontaneous resolution of pelvic retroversion and thoracic hypokyphosis.^{92,93}

The role of musculature in supporting lumbar lordosis is clearly established through observations of human-specific evolutionary changes in spinal musculature, the mechanical role of spinal muscles, and the correlations between muscle volume and postural changes. However, clinical research has not yet established a cause or effect relationship between spinal muscles and postural changes. Establishing a timeline for degenerative postural changes and musculature may provide insights into the effectiveness and timing of muscle development rehabilitation strategies. Effective conservative treatments also depend on identifying patients at risk for postural degeneration before they become symptomatic. Recent research to establish definitions of normal lumbar curvature and optimize lordosis measurement methods²⁵ provides an important foundation to establish early diagnosis and track postural degeneration. The relationship between vertebral structure and posture defined for studying human evolution may provide a means for early identification of small deviations from an individual's optimal posture. These structure-postural relations may also be able to predict the degree of spontaneous correction possible in the thoracic spine following the restoration of lumbar lordosis.

Lumbar flattening often results from degenerative changes in the spine. Osteoporotic wedge fractures in the vertebrae⁸¹ and degenerative disc disease¹⁶ both correlated with reduced lordosis in the lumbar spine. Muscle weakness also correlated with a loss of lordosis. Older patients show a reduction in lumbar lordosis as a result of these degenerative changes.^{75,143} However, the degree of lumbar lordosis in normal individuals is highly variable (41°–70°)^{23,194} and broadly overlaps with measures of degenerative hypolordosis (–40° to 67°).^{31,99} Therefore, defining threshold values for degenerative lumbar curvature in isolation is meaningless. Recently, Schwab et al.¹⁷³ defined postural deformity criteria for the spine. Lumbar spine deformity is determined by the difference between a patient's lumbar lordosis (measured by Cobb angle) and PI ($LL = PI \pm 9^\circ$). These deformity criteria closely correlate with health-related quality of life measures.¹⁸⁷

Radiographic metrics play an important role in diagnosing sagittal alignment and guiding surgical planning.⁸ The standard modality for assessing sagittal alignment is the lateral 36-in standing radiograph. Although a patient's deformity may be concentrated in the lumbar spine, diagnosing postural deformity includes consideration of the entire spinopelvic structure. The sagittal vertical axis (SVA) is the most common global measure of sagittal alignment and is defined as the horizontal offset between the C-7 plumb line and posterior superior aspect of the S-1 vertebral body. The pelvis is a significant contributor to sagittal alignment and the PI, pelvic tilt, and sacral slope are also measured (Fig. 1).^{109,141} Lumbar lordosis in healthy individuals is highly correlated with both pelvic incidence (0.68) and sacral slope (–0.76).¹⁹⁴ However, PI is a fixed measure,

independent of standing position, whereas sacral slope may be affected by pelvic retroversion and knee flexion typical of patients with significant loss of lumbar lordosis.^{104,171} A patient's PI assists in determining the degree of total lumbar lordosis required to achieve better sagittal alignment; however, total spine correction can be obtained from an infinite combination of local curves (Fig. 3). Research on normal spines demonstrates a consistent pattern of curvature distribution in the lumbar spine, with the L5–S1 level contributing 40% of the total lumbar lordosis.²² However, there is significant variation in the pattern. Further research is required to determine the effect of curvature distribution in the lumbar spine on patient outcome and to develop methods to define the optimal distribution of lordosis in the fixed lumbar spine.

The goal of treating lumbar deformity is to improve a patient's quality of life. Lumbar deformity may be treated conservatively or surgically. Patients with mild to moderate symptoms may be treated with bracing, pain control, and physical therapy; however, none of these are curative and at best prevent the progression of a deformity. Conservative management is more effective in patients with flexible deformities than in those with fixed deformities.¹⁶¹ Patients managed nonoperatively have shown increased use of narcotics, epidural blocks, analgesics, pain management referrals, and bed rest.⁶⁶ In cases of degenerative disease, a small number of patients benefit from spinal steroid injections to improve pain and function; however, more than two-thirds require invasive treatment within 2 years.⁴⁰ Although the cost of spinal surgery exceeds that of nonsurgical management, treatment effects significantly favor surgery.⁶² There are disadvantages to surgery, particularly the loss of flexibility involved in pelvic fusion and peri- and postoperative complications. In addition, not all patients are viable candidates because of medical comorbidities or personal preference. However, the advantages of surgical correction of sagittal malalignment and the improvements in health-related quality of life measures are widely recognized.^{67,172}

Fig. 3. Illustration showing the variation in lumbar lordosis (LL). The Cobb angle is a standard measure of lumbar lordosis and is the angle between the sacral plate and the superior surface of the L-1 vertebra. Although patients may present with the same measure of lumbar lordosis, the relative angle of each vertebra in the lumbar spine can vary, giving the surgeon multiple options for the curvature of spinal fusion. There is currently no protocol for optimizing the distribution of curvature correction in the lumbar spine to an individual patient. Copyright Andrew McNichol. Published with permission.

The goals of surgical treatment are restoration of proper spinal alignment and alleviation of symptoms. Surgery should strive to achieve an SVA less than 5 cm and pelvic tilt less than 20°, while alleviating PI/lumbar lordosis mismatch (that is, $PI = \text{lumbar lordosis} \pm 9^\circ$).¹⁷³ Ideal alignment allows minimal muscle expenditure while standing, reflected in the “Cone of Economy” principle described by Jean Dubousset (Fig. 2).⁵⁴ However, surgically fixed lumbar lordosis may result in increased muscle activity in seated postures, where a normal lumbar spine would flatten and reduce extensor muscle activity. Mechanical studies of spine posture demonstrate flexion relaxation in the extensor muscles during relaxed seated postures. Surgical fixation with constant lumbar lordosis may inhibit the flexion relaxation phenomenon. Further study is needed to determine the energetic costs of surgically fixed lumbar lordosis in nonstanding activities.

Since global sagittal malalignment correlates with health-related quality of life scores for patients with spinal deformity, achieving appropriate postoperative alignment is critical. Improved recognition of this relationship has led to increased use of multilevel fusions and posterior osteotomies. Spinal osteotomies allow for correction of rigid deformity patterns and can be applied to a variety of pathologies including adult scoliosis, flatback syndrome, iatrogenic fixed sagittal imbalance, kyphotic decompensation syndrome, and flat buttock.¹⁷⁴ Multilevel fusions can lead to adjacent level degeneration^{56,72} and coronal imbalance¹⁴⁸ when not performed optimally. Adjacent level effects are significantly reduced in patients with a normal postoperative C-7 plumbline¹⁰² and in lumbar spine fusions that extended above T-10.¹⁰⁰

Outcomes in spinal deformity surgery vary by the procedure type, level involvement, and patient demographic. Although there is debate over the optimal management of symptomatic spinal deformity, surgical treatment is associated with improved outcomes compared with conservative nonoperative management.^{37,180,181} Since sagittal plane malalignment is the main radiographic driver of disability in these patients,^{67,104} correction of spinopelvic parameters is critical. Restoration of sagittal alignment is associated with improved postoperative outcomes and fewer long-term complications.^{89,100} However, surgical corrections of postural deformities have high complication rates.^{3,152} Complications are significantly increased by advanced age,^{43,58,129} medical comorbidities,^{15,192} nutritional status,¹⁰⁶ length of fusion,⁴³ and extension of fusion to the sacrum.³⁶ The history of previous surgery has been shown to increase complication rates in some studies⁶⁸ but not others.¹¹⁵ Women also experience more complications following spinal fusion than men, even after accounting for osteoporosis.⁵⁸ This may be due to sexual dimorphism in the structure of the spine and vertebrae as observed through studies of human evolution. Further research is required to determine any differences in diagnosis and treatment of postural deformity based on dimorphic structures in the spine. Among adults undergoing surgical correction of sagittal plane deformity, the best health-related quality of life outcomes are achieved in patients who undergo substantial correction of SVA, defined as greater than 120 mm and at least 66%.³⁴ Although lower amounts of correction can still yield clinical

improvements, the minimal clinically important difference in health-related quality of life scores was not significantly different for such mild or moderate degrees of correction, stressing the need for complete sagittal plane correction to achieve the best clinical outcomes. Surgical correction of a severe spinal deformity is complex, risky, and expensive. Identifying patients who are at risk for postural deformity and developing methods for monitoring and treating patients early will reduce the need for these invasive surgeries and improve patient quality of life.

Mechanics, biology, and evolution all point to the need for a stable and flexible spine to perform normal daily activities. Dynamic spine constructs propose to accomplish stabilization while maintaining flexibility after surgery; however, current systems have been plagued by complications and failures.^{90,121} Current dynamic systems are not suitable for multilevel postural corrections.⁹ An effective dynamic system should help to preserve muscle strength and bone health by allowing the dynamic motion for which the human body evolved. A dynamic fixation system suitable for postural deformities could significantly improve patient quality of life by reducing the energetic costs of fixed lordosis and reduce complications by eliminating load and deformation concentrations in the spinal levels above and below the fusion instrumentation. An effective method for restoring spine posture while maintaining flexibility remains an elusive goal. Future research should aim to develop new dynamic fixation tailored to spinal deformity treatment. In addition, more research is needed on the effect of rigid fixation on patient activities, energetic costs, and postural compensation strategies for large ROM activities such as sitting or bending.

Conclusions

The evolution and development of human lumbar lordosis highlight the interdependence of pelvic structure and lumbar lordosis. Lumbar lordosis is dictated by pelvic incidence, spinal musculature, vertebral wedging, and disc health. A change in any of these factors affects the distribution of mechanical loading in the spine. Disc degeneration and osteoporosis are strongly influenced by genetics, with limited effects of loading or environment. Therefore, postural degeneration is not simply a result of misusing our evolved spines. Spinal musculature shows a strong correlation with lumbar lordosis and may provide an opportunity for early intervention or prevention of postural deformities. Developing tools for early identification of patients at risk for postural deformities through patient history (genetics, mechanics, and environmental exposure) and tracking postural changes over time should reduce the need for complex and costly spinal fixation.

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