Biological Theory
https://doi.org/10.1007/s13752-022-00402-4

ORIGINAL ARTICLE

Acquired Spinal Conditions in Evolutionary Perspective: Updating a Classic Hypothesis

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Received: 14 April 2021 / Accepted: 18 March 2022

Abstract
In 1923, Sir Arthur Keith proposed that many common back problems are due to the stresses caused by our evolutionarily novel form of locomotion, bipedalism. In this article, we introduce an updated version of Keith's hypothesis with a focus on acquired spinal conditions. We begin by outlining the main ways in which the human spine differs from those of our closest living relatives, the great apes. We then review evidence suggesting there is a link between spinal and vertebral shape on the one hand and acquired spinal conditions on the other. Next, we discuss recent studies that not only indicate that two common acquired spinal conditions—intervertebral disc herniation and spondylolysis—are associated with vertebral shape, but also suggest that the pathology-prone vertebral shapes can be understood in terms of the shift from quadrupedalism to bipedalism in the course of human evolution. Subsequently, we place the aforementioned findings under an umbrella hypothesis, which we call the "Evolutionary Shape Hypothesis." This hypothesis contends that individuals differ in their propensity to develop different acquired spinal conditions because of differences in vertebral shape that relate to the evolutionary history of our species. We end the article with some possible directions for future research.

Keywords 3D geometric morphometrics · Acquired spinal condition · Back pain · Darwinian medicine · Evolutionary medicine · Human evolution · Intervertebral disc herniation · Palaeopathology · Spondylolysis · Vertebral shape

Introduction

Back pain's importance is hard to overstate. Surveys indicate that it is experienced by as many as two-thirds of people at some point in their lives, making it one of the commonest health problems (Webb et al. 2003; Hoy et al. 2014). It is also one of the most impactful. Currently, it is the greatest contributor to disability worldwide (Maher et al. 2017). Because of its prevalence and the fact that it is often debilitating, back pain has substantial economic impacts. For instance, it has been estimated to cost the US as much as $90 billion in direct and indirect costs (Davis 2012). The equivalent figures for Australia and the UK are >$9 billion per year and £12 billion per year, respectively (Maniadakis and Gray 2000; Walker et al. 2003; Donaldson 2008). To take a fourth example, the direct and indirect costs of back pain in Canada have been estimated to exceed $12 billion per annum (Bone and Joint Canada 2014). Needless to say, given the individual and societal impacts of back pain, improving understanding of its causes is an important task for researchers.

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Published online: 06 July 2022

A major hurdle in the prevention and treatment of back pain is our limited understanding of why, within a group of ostensibly similar people (i.e., same sex, age, ethnicity, etc.), some individuals suffer from back pain while others do not. Another substantial hurdle is the complex and multifactorial etiology of many spinal conditions. Clinical studies have identified associations with a number of potential etiological factors, including genetics, diet, activity, and biochemistry, but few of these associations have been confirmed by subsequent studies (e.g., Adams and Roughly 2006; Nuckley et al. 2008; Hackinger et al. 2017). In fact, to date, the only factor consistently linked to a future episode of back pain is a history of back pain (Stanton et al. 2008).

Back pain is a complex phenomenon. It can occur in any of the four regions of the spine, i.e., the cervical region, the thoracic region, the lumbar region, or the sacral region (Webb et al. 2003). It can be chronic or acute (Hoy et al. 2014). It can be congenital (present at birth regardless of cause), acquired (developed during life as a result of degeneration or trauma), or idiopathic (no known cause) (Adams and Roughly 2006; Nuckley et al. 2008; Stanton et al. 2008; Hackinger et al. 2017; Maher et al. 2017). And it can involve soft tissue, bone, or both (Maher et al. 2017). In this article, we focus on acquired spinal conditions, which are thought to be among the most common causes of back pain (Amirdelfan et al. 2014).

Humans experience acquired spinal conditions far more frequently than nonhuman apes (Jurmain 1989; Lovell 1990; Filler 2007; Lowenstine et al. 2016). For example, arthritis of the vertebral bodies, which is also known as spondylosis, has been found to occur in about 76% of modern humans (Muraki et al. 2009). In contrast, spondylosis affects only 4% of gorillas, 5% of bonobos, and 2% of chimpanzees (Jurmain 2000). Likewise, spondylolysis, which is a cleft in the neural arch that is caused by a fatigue fracture at the site of the pars interarticularis (Merbs 1996; Mays 2006, 2007; Hu et al. 2008), is relatively common among humans, especially in the lower lumbar spine (May et al. 2006; Hu et al. 2008), but is not known to occur in great apes (Merbs 1989, 1996; Ward and Latimer 2005). The situation is similar for intervertebral disc herniation, which is a condition where the gel-like substance inside the intervertebral disc, the nucleus pulposus, prolapses through the fibrous layers of the disc (Hickey and Hukins 1980). When the results of studies that have assessed the frequency of skeletal markers of intervertebral disc herniation in humans and nonhuman apes are compared (Lovell 1990; Dar et al. 2009), it is clear that modern humans suffer from intervertebral disc herniation far more frequently than do great apes. Dar et al. (2009) found that 48% of their modern human specimens exhibited evidence of intervertebral disc hernias, whereas Lovell (1990) discovered that only 2% of the great ape vertebrae in her sample had such evidence.

It is possible that the much higher frequency of occurrence of some acquired spinal conditions in humans compared to great apes is due to our greater average lifespan. This may be the case for spondylosis, which has been found to increase in frequency and severity with age in *Homo sapiens* (Middleton and Fish 2009; Molnar et al. 2009). However, not all of the differences between humans and great apes in the frequency of occurrence of acquired spinal conditions can be explained in this way. Intervertebral disc herniation and spondylolysis, for example, tend to affect humans at a relatively young age and have not been found to correlate strongly with increasing age (Pfrirrmann and Resnick 2001; Burke 2012). So, it is unlikely that the greater average lifespan of *H. sapiens* explains the difference in the frequency with which humans and great apes exhibit these conditions. Average life span may play a role, but it is clearly not the major factor.

It has long been suspected that the stress that bipedalism puts on our spines, most notably vertical compressive loading, is an important etiological factor for the acquired spinal conditions that afflict our species. This hypothesis was first proposed by the famous Scottish anatomist and anthropologist Sir Arthur Keith, who outlined it in a series of lectures that were delivered at The Royal College of Surgeons of England and later published in the *British Medical Journal* (Keith 1923). It has since been supported by many other researchers, including Krogman (1951), Merbs (1996), Jurmain (2000), Latimer (2005), Filler (2007), Plomp et al. (2015), and Been et al. (2019).

A number of empirical studies published in the last 20 years have investigated the hypothesized relationship between bipedalism and acquired spinal conditions (e.g., Scannell and McGill 2003; Ward and Latimer 2005; Masharawi et al. 2007; Ward et al. 2007; Meakin et al. 2008, 2009; Masharawi 2012; Plomp et al. 2015, 2019a, 2020; Meyer 2016; Been et al. 2019). Collectively, these studies suggest that the relationship is mediated by the nature of the curvature of the spine (Meakin et al. 2008; Been et al. 2019). They also suggest that the relationship is influenced by characteristics of the individual vertebrae (Scannell and McGill 2003; Ward and Latimer 2005; Masharawi et al. 2007; Ward et al. 2007, 2010; Meakin et al. 2009; Masharawi 2012; Plomp et al. 2015, 2019a, 2020; Meyer 2016). The lumbar vertebrae are particularly important in this regard. The reason for this is that the incidence of acquired spinal conditions is much higher in the lumbar region of the spine than in the cervical and thoracic regions (Battie et al. 2009; Sparrey et al. 2014), a fact that has led the lumbar region to be called "the evolutionary weak point" of the human spine (Sparrey et al. 2014, pp. 4).

The goal of this article is to introduce an updated version of Keith's (1923) hypothesis, with a focus on acquired spinal conditions. The article is structured as follows. In the next section, we explain how the shape of the spine and lumbar vertebrae relate to bipedal posture and locomotion. We concentrate on the lumbar vertebrae not only because the shape of the lumbar region is particularly important for bipedalism, but also because, as we explained earlier, acquired conditions are more common in the lumbar region than in the other regions. Subsequently, we discuss clinical and comparative evidence that indicates there is an association between acquired spinal conditions and the shape of the lumbar spine and its constituent vertebrae. Thereafter, we outline recent studies that suggest the shapes associated with different acquired spinal conditions can be understood in evolutionary terms. In the fifth section, we outline our version of Keith's (1923) hypothesis, which we call the "Evolutionary Shape Hypothesis." In the final section of the article, we suggest some potential future research directions.

Adaptations for Bipedalism in the Human Lumbar Spine

When the human spine is considered as an anatomical unit, there are two main features that are thought to be adaptations for bipedal posture and gait. One is its distinctive pattern of curvature. While great apes have a roughly C-shaped spine, healthy adult humans have a sinuous spine (Fig. 1). This shape is a consequence of the four spinal regions being curved in different directions (Keith 1923; Latimer and Ward 1993; Shapiro 1993; Ward and Latimer 2005; Been et al. 2010).

The cervical region of the human spine exhibits lordosis, which is a forward curve. This results from the intervertebral discs being dorsally wedged, i.e., shorter at their dorsal border than at their ventral border (Been et al. 2010). In contrast, the thoracic region exhibits kyphosis, which is a backward curve. This is due to ventral wedging of the vertebral bodies, i.e., shorter at their ventral border than at their dorsal border (Latimer and Ward 1993). The lumbar region, like the cervical region, exhibits lordosis. Unlike in the cervical region, however, the lordosis of the lumbar region is facilitated by dorsal wedging of the intervertebral discs *and* vertebral bodies (Been et al. 2010). The sacral region of the spinal column has a kyphotic curve. This curve results from ventral wedging of the second to fifth sacral vertebrae and the coccygeal vertebrae and is enhanced by a ventral tilt of the cranial end of the sacrum (Cheng and Song 2003). While the kyphoses of the thoracic and sacral regions appear early in fetal development, the cervical and lumbar lordoses continue to develop until about 13 years of age (Okpala 2016). The four curves of the human spine are widely accepted to be important for bipedalism (Latimer and Ward 1993; Been et al. 2010). They bring the center of gravity of the body over the hips, and therefore allow the trunk to be balanced above the legs during bipedal walking (Latimer and Ward 1993; Been et al. 2019). The lumbar curve is particularly significant in this regard (Latimer and Ward 1993; Been et al. 2010, 2019).

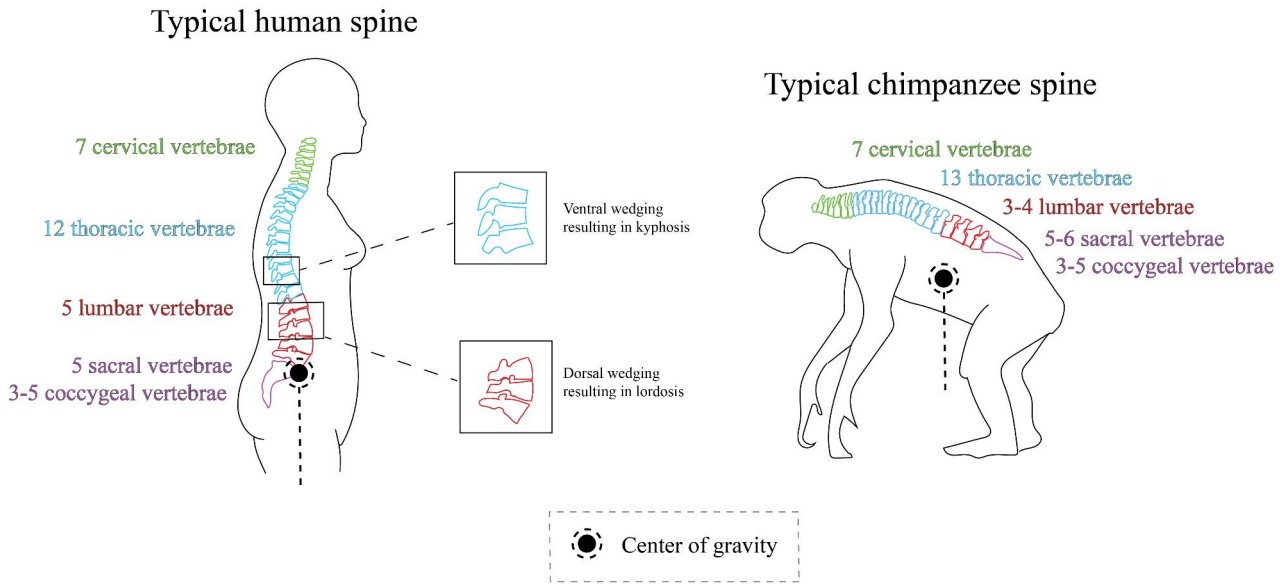

Fig. 1 Drawing comparing the human spine and the chimpanzee spine

The other major feature of the human spine that is thought to be an adaptation for bipedalism is its vertebral formula, i.e., the most common number of vertebrae in the four regions (Fig. 1) (Williams 2012). Individuals of all hominoid species usually have seven cervical vertebrae, but there is variation in the modal number of thoracic, lumbar, and sacral vertebrae among species. Humans generally have 12 thoracic, five lumbar, five sacral, and three to five coccygeal vertebrae (Williams 2012). Chimpanzees, bonobos, and gorillas typically have 13 thoracic, three to four lumbar, five to six sacral, and three to five coccygeal vertebrae, while the equivalent figures for bonobos are 13–14, 3–4, 6–7, and 3–5, respectively (Williams 2012). Orangutans usually have 12 thoracic vertebrae, four lumbar vertebrae, five sacral, and four to six coccygeal vertebrae (Williams 2012). Thus, humans tend to have a longer lumbar region than the other hominoids. This has been argued to result in an increased range of motion for flexion and extension (Bramble and Lieberman 2004; Williams 2012). Additionally, it has been proposed that the larger gap between the ribcage and the iliac blades created by the longer lumbar spine allows for counter-rotation of the trunk relative to the hips, which helps to maintain balance during bipedal locomotion (Bramble and Lieberman 2004).

Turning now to the lumbar vertebrae, many of the traits that distinguish those of humans from those of the great apes appear to relate to facilitating and maintaining lumbar lordosis (Fig. 2). For example, the orientation of the zygapophyseal facets is thought to be linked to vertebral slippage (i.e., horizontal movement of the vertebra away from its normal location) and rotation in the context of posture and gait (Latimer and Ward 1993; Shapiro 1993). All spines allow for some rotation, and some slippage of vertebrae is bound to occur, but too much of either would cause instability in the spine and potentially impact the soft tissues associated with the vertebrae, including the spinal cord. In great apes, the facets of the upper lumbar vertebrae are obliquely oriented, while in humans these facets are oriented more towards the sagittal plane, which has been hypothesized to resist rotation and maintain lumbar lordosis (Latimer and Ward 1993; Shapiro 1993; Been et al. 2010). The pattern changes in the final two lumbar vertebrae. In humans, the facets of the fourth and fifth lumbar vertebrae become more coronally oriented, likely to resist ventral slippage.

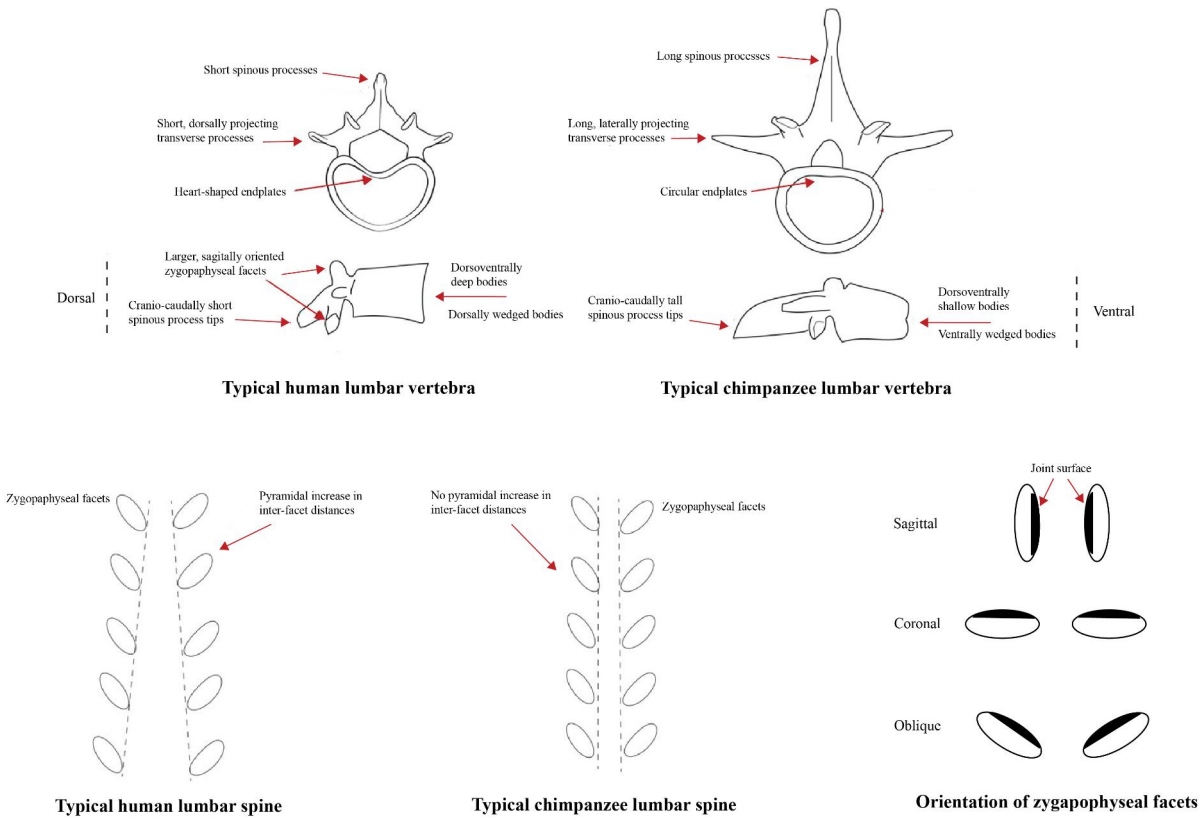

Fig. 2 Simplified drawing illustrating the main shape differences between a typical human lumbar vertebra and a typical chimpanzee lumbar vertebra

Conversely, the facets of the last two lumbar vertebrae in great apes become more sagittally oriented compared to the facets in their upper lumbar vertebrae (Latimer and Ward 1993). In addition, as Fig. 2 indicates, in humans the distance between the zygapophyseal facets gradually increases as one moves down the lumbar spine (Latimer and Ward 1993). This has been suggested to provide sufficient spacing between the facets of subjacent vertebrae so that they do not impinge upon each other due to lumbar lordosis (Ward and Latimer 2005; Ward et al. 2007, 2010).

The form of the lumbar transverse processes may also play an important role in maintaining lumbar lordosis. In particular, the transverse processes of human lumbar vertebrae are shorter and more dorsally orientated than those of the great apes (Latimer and Ward 1993; Cheng and Song 2003). Usually referred to as "invagination" of the vertebral column (Latimer and Ward 1993), the dorsal projection of the transverse processes positions the spine forward in the thorax (Bogduk et al. 1992; Shapiro 1993; Been et al. 2010, 2019). This increases the length of the lever arms of the epaxial muscles (i.e., the dorsal muscles of the thorax) and therefore improves their ability to extend the spine into an upright posture, resist lateral flexion and anterior shear force, and maintain lumbar lordosis during bipedal posture and gait (Bogduk et al. 1992; Shapiro 1993; Sparrey et al. 2014).

Several traits that distinguish the spinous processes of human lumbar vertebrae from those of great apes have likewise been argued to facilitate lumbar lordosis. In particular, the spinous processes of human lumbar vertebrae are dorsoventrally shorter (Bogduk et al. 1992; Latimer and Ward 1993) and have craniocaudally pinched tips (Plomp et al. 2019b). The relative shortness of the spinous processes has been hypothesized to decrease the lever arms of the spinal extensor muscles and therefore limit the degree of sagittal mobility of the spine (Bogduk et al. 1992; Ward and Latimer 2005). The craniocaudal pinching of the processes' tips has been suggested to facilitate lumbar lordosis by increasing the spacing between the spinous processes of subjacent vertebrae (Shapiro 1993; Plomp et al. 2019b).

There are four other traits that differentiate the human lumbar spine from that of the great apes. First, the bodies of human lumbar vertebrae are dorsoventrally deeper than those of great apes (Latimer and Ward 1993; Plomp et al. 2015). Second, the endplates of the human lumbar vertebrae are more heart-shaped than those of great apes (Robinson 1972; Plomp et al. 2015). Third, the vertebral bodies gradually increase in mediolateral width as one moves down the human lumbar spine (Rose 1975). Lastly, the pedicles of the last two lumbar vertebrae in the human spine are mediolaterally wider than those of the great apes (Shapiro 1993). All four of these traits have been hypothesized to help the vertebrae withstand the compressive load acting on the lower spine (Rose 1975; Latimer and Ward 1993; Been et al. 2010; Plomp et al. 2015, 2019b).

Evidence for an Impact of Spinal and Vertebral Shape on Spinal Health

Many of the studies that have investigated the relationship between vertebral shape and spinal health have focused on lumbar lordosis (e.g., Scannell and McGill 2003; Keller et al. 2005; Been and Kalichman 2014; Been et al. 2019; Zlolniski et al. 2019). The lordotic angle has been particularly important in these studies. Measured between a line running parallel to the superior endplate of the first lumbar vertebrae and a line running parallel to the sacral endplate, this angle is associated with lumbar lordosis such that a large lordotic angle corresponds to a more pronounced lumbar lordosis, whereas a small lordotic angle equals a less pronounced lumbar lordosis. The size of the lordotic angle is highly variable in *H. sapiens* (Been and Kalichman 2014; Zlolniski et al. 2019). The average lordotic angle is estimated to be between 51–53° (Been et al. 2010; Yang et al. 2014), while an angle ranging from 57° to 75° is considered pronounced (Been et al. 2019), and an angle of 40° or less is deemed small (Endo et al. 2010; Sak et al. 2011; Yang et al. 2014). This variation is associated with the propensity to develop acquired spinal diseases (Scannell and McGill 2003; Keller et al. 2005; Been et al. 2019).

One acquired spinal disease that has been linked with the lordotic angle is osteoarthritis of the zygapophyseal joints. Osteoarthritis is a breakdown of synovial joints, which are the moveable joints of the body. In the spine, there are two types of synovial joints—the zygapophyseal joints, which link the articular processes of two adjacent vertebrae, and the costovertebral joints, which link the ribs to the thoracic vertebrae. Osteoarthritis particularly affects the zygapophyseal joints. Symptoms of zygapophyseal joint osteoarthritis include localized tenderness and pain (Dolan et al. 1996), which usually worsens with spinal extension, sitting, or standing (Dolan et al. 1996; Borenstein 2004). Clinically, zygapophyseal osteoarthritis preferentially affects individuals with pronounced lumbar lordosis (Roussouly and Pinheiro-Franco 2011). Its occurrence in the lumbar spine also seems to correlate with zygapophyseal facets that are more sagittally oriented than in healthy individuals (Fujiwara et al. 2001). Based on these clinical findings, researchers have proposed that a more-pronounced-than-normal lumbar lordosis results in both increased contact between the vertebral facets and a greater amount of shear force acting on the joints, and that this increases the likelihood of the joints

breaking down and developing osteoarthritis (Roussouly and Pinheiro-Franco 2011; Weinberg et al. 2017).

Spondylolysis has also been correlated with a more-pronounced-than-normal lumbar lordosis. To reiterate, spondylolysis is a cleft in the neural arch that is caused by a fatigue fracture at the site of the pars interarticularis (Merbs 1996; Mays 2006, 2007; Hu et al. 2008). People who play a lot of sports have been found to be particularly prone to develop spondylolysis (Iwamoto et al. 2004), with nearly 50% of adolescent athletes who report low back pain being subsequently diagnosed with the condition (Micheli and Wood 1995). In addition, bilateral spondylolysis can result in a loss of the anchoring effects of the zygapophyseal facets, causing the vertebral body to slip forward in the spine. When this occurs, the condition is called spondylolisthesis (Rossi and Dragoni 2001).

Several studies have linked spondylolysis with greater than normal lumbar lordosis. Using clinical radiographs, Roussouly et al. (2006) found that spondylolysis is associated with increased lordosis in a sample of living humans, and hypothesized that a more-pronounced-than-normal lumbar lordosis increases direct contact between the neural arches of the lumbar vertebrae and eventually causes the fractures that lead to spondylolysis. Subsequently, Masharawi (2012) discovered that lumbar vertebrae with spondylolysis tend to have vertebral bodies that are more dorsally wedged than healthy vertebrae. This is consistent with Roussouly et al.'s (2006) findings because greater dorsal wedging of the lumbar vertebrae facilitates a more pronounced lumbar lordosis (Been et al. 2010). Other research teams have also found that the facets of the L4 and L5 vertebrae of individuals with spondylolysis tend to be flatter, more coronally oriented, and smaller in the transverse direction than those of individuals without spondylolysis (Grobler et al. 1993; Miyake et al. 1996; Van Roy et al. 2006). As we alluded to earlier, the shape and orientation of the vertebral facets are associated with the curvature of the spine (Shapiro 1993). In the lumbar spine, the zygapophyseal facets are oriented towards the sagittal plane, which likely helps to resist rotation and maintain lumbar lordosis (Ahmed et al. 1990; Shapiro 1993; Been et al. 2010, Jaumard et al. 2011). Based on this, it has been suggested that the flatness and coronal orientation of the facets identified in L4 and L5 vertebrae with spondylolysis may not provide adequate support for the large lordotic angle that is also associated with the lesion (Plomp et al. 2020).

While a number of studies suggest that having a pronounced lordotic angle may increase the likelihood of developing zygapophyseal osteoarthritis and spondylolysis, there is also evidence that having a smaller-than-normal lordosis may negatively impact an individual's spinal health. Several papers have reported that people with evidence of degenerative disc disease and intervertebral disc herniation have significantly smaller lordotic angles than those with healthy spines (Barrey et al. 2007; Ergun et al. 2010; Yang et al. 2014). The studies in question have found that individuals with degenerative changes to their discs have an average lordotic angle of 40° while those with disc herniations have an average lumbar lordosis angle of 37° (Endo et al. 2010; Sak et al. 2011; Yang et al. 2014). Both of these angles are considerably smaller than the average lumbar lordosis angle for individuals with healthy lumbar spines.

Three other traits have been found to correlate with intervertebral disc herniation in modern humans. One of these traits was identified by Harrington et al. (2001). These authors used CT scans of 97 patients to measure vertebral endplate dimensions and found that individuals with herniated intervertebral discs tended to have endplates that are more circular in shape. This finding was confirmed by Plomp et al. (2012), who compared the two-dimensional (2D) shape of vertebrae in skeletons with and without Schmorl's nodes, which are depressions on the vertebral endplate formed by a herniated disc (Schmorl and Junghanns 1971), and found that vertebrae with Schmorl's nodes tend to have more circular vertebral bodies. Another one of the traits was recognized by Pfirrmann and Resnick (2001). These authors performed an analysis of thoracic and lumbar vertebrae and intervertebral discs from 128 cadavers and discovered that intervertebral disc herniations affected vertebrae with flatter endplates significantly more frequently than vertebrae with more concave endplates. The third trait was identified by Plomp et al. (2012). It is relatively short pedicles.

Evolutionary Shape Variation and Spinal Health

The growing evidence that spinal and vertebral shape influences an individual's propensity to develop acquired spinal conditions raises the question of why some people have spinal and vertebral shapes that predispose them to such conditions while others do not. Recently, several studies have attempted to answer this question from an evolutionary perspective.

Plomp et al. (2015) used 2D shape data to compare the shape of human vertebrae with and without Schmorl's nodes to those of chimpanzees and orangutans. They found that human vertebrae with Schmorl's nodes are more similar in shape to the vertebrae of chimpanzees than are healthy human vertebrae. Specifically, both human vertebrae with Schmorl's nodes and chimpanzee vertebrae tend to have more circular vertebral bodies and relatively shorter pedicles than healthy human vertebrae (Plomp et al. 2015). Because there is general agreement that *Homo* and *Pan* share an

exclusive common ancestor and that this ancestor was quadrupedal, Plomp et al. (2015) proposed that individuals who develop intervertebral disc hernias do so because their vertebrae fall at the ancestral end of the range of variation in humans and therefore are less well adapted for the stresses associated with bipedalism. They called this the "Ancestral Shape Hypothesis."

Subsequently, Plomp et al. (2019) tested the Ancestral Shape Hypothesis with three-dimensional (3D) shape data from the last two thoracic and first lumbar vertebrae of pathological humans, healthy humans, chimpanzees, and several fossil hominin species. They were able to confirm that Schmorl's nodes-affected and healthy human vertebrae differ significantly in shape, and that Schmorl's nodes-affected human vertebrae are closer in shape to those of chimpanzees than are healthy human vertebrae. Additionally, they found that pathological human vertebrae are generally more similar in shape to the vertebrae of the fossil hominins than are healthy human vertebrae, which is also consistent with the Ancestral Shape Hypothesis. According to Plomp et al.'s (2019) results, Schmorl's nodes-bearing human vertebrae tend to have vertebral bodies that are more circular and more ventrally wedged, implying a smaller lordotic angle; relatively short pedicles and laminae; relatively long, more cranio-laterally projecting transverse processes; and relatively long, cranially-oriented spinous processes (Fig. 3).

Most recently, Plomp et al. (2020) investigated the evolutionary basis of spondylolysis. As noted earlier, individuals with spondylolysis have been found to have more-pronounced-than-normal lumbar lordosis (Masharawi 2012). Building on this association, Plomp et al. (2020) hypothesized that spondylolytic vertebrae have the converse shape problem to those with Schmorl's nodes, i.e., they exhibit shape traits that are exaggerated adaptations for bipedalism. To test this "Overshoot Hypothesis," they compared the 3D shape of final lumbar vertebrae of humans, chimpanzees, gorillas, and orangutans. The humans were divided into three groups according to whether they had bilateral spondylolysis, Schmorl's nodes on any vertebrae, or no vertebral lesions. Consistent with the predictions of the hypothesis, Plomp et al. (2020) found that spondylolytic human vertebrae shared fewer shape similarities with great ape vertebrae than did the healthy human vertebrae. They also found that the vertebrae of humans with Schmorl's nodes had more similarities in shape with great ape vertebrae than did either spondylolytic or healthy human vertebrae. Since the spondylolytic vertebrae were farthest from great ape vertebrae in terms of shape, Plomp et al. (2020) concluded that spondylolysis is indeed partly the result of individuals having exaggerated vertebral adaptations for bipedalism.

The Evolutionary Shape Hypothesis

A few years ago, Crespi and Go (2015) outlined what they called the "Diametrical Disease Framework" for understanding psychiatric, rheumatological, neurological, oncological,

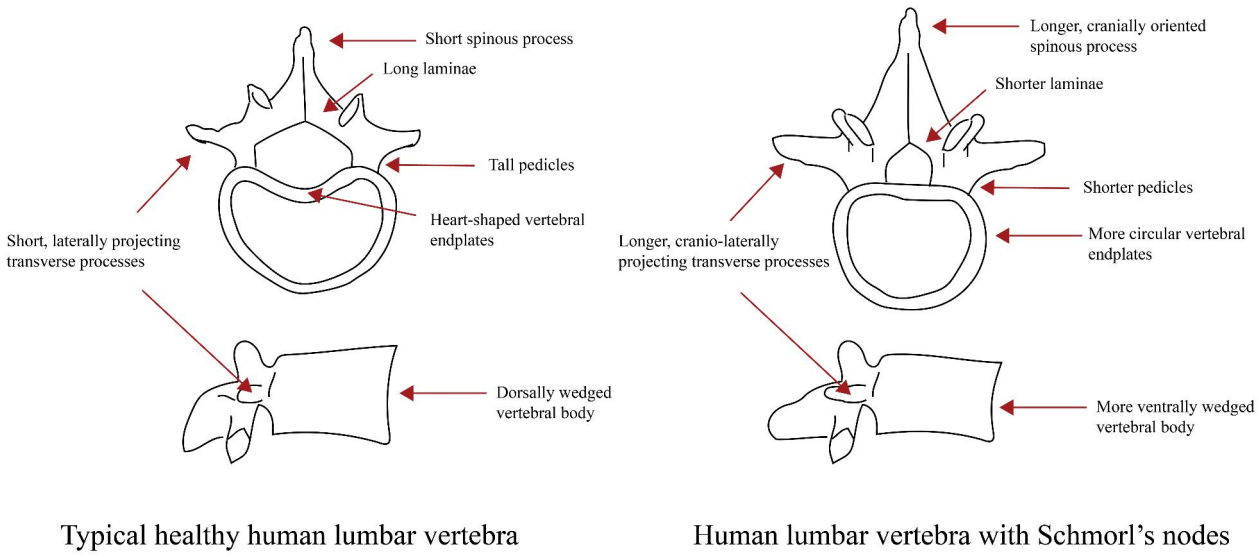

Fig. 3 Simplified drawing depicting the shape differences between a typical healthy human lumbar vertebra and a human lumbar vertebra with Schmorl's nodes

and immunological conditions. They argued that it can be helpful to think about health conditions in terms of trade-offs, where an increased risk of one condition can decrease the risk of another condition and vice versa. When combined with Plomp et al.'s (2015, 2019, 2020) results, this framework enables us to update Keith's (1923) idea that bipedalism predisposes us to acquired spine conditions.

We can conceptualize the distribution of vertebral shape variation in humans as a bell curve with an ancestral end and a derived end (Fig. 4). At the center of the range of variation are vertebrae that have the lineage-specific optimal shape for bipedalism and, therefore, have a lower probability of developing spinal pathologies in response to the stresses of bipedal posture and gait (we use the term "lineage-specific optimal shape" because natural selection is constrained by history and therefore is not expected to produce globally optimal solutions; Gould and Lewontin 1979; Beatty and Desjardins 2009). At the ancestral end of the range, vertebrae differ little from those of the chimpanzees and, by extension, from those of the common ancestor of humans and chimpanzees. People with vertebrae that fall in this part of the distribution have a heightened probability of developing intervertebral disc hernias. Conversely, at the other, highly derived end of the range of shape variation, individuals exhibit exaggerated versions of our species' vertebral adaptations for bipedalism. Individuals with vertebrae that

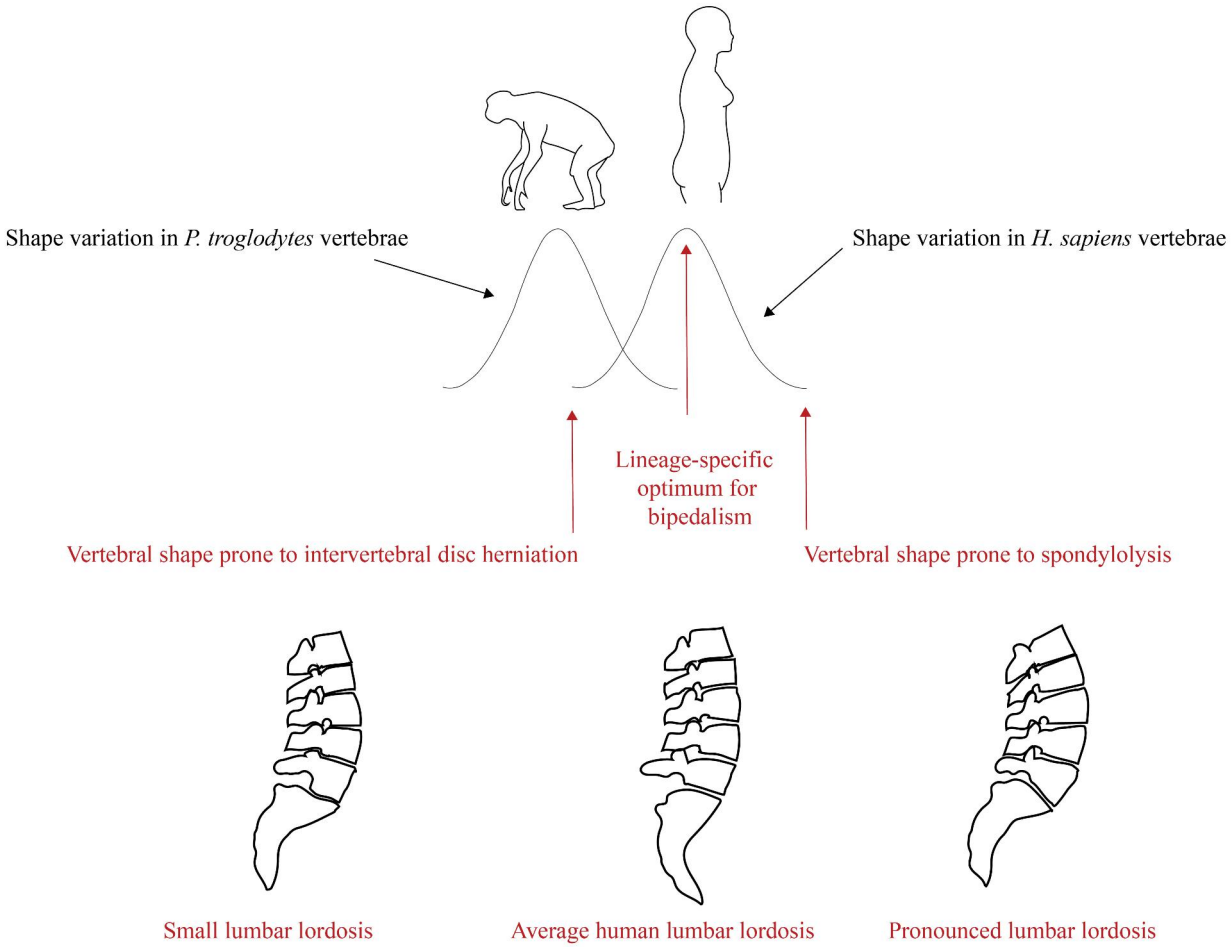

Fig. 4 The logic of the Evolutionary Shape Hypothesis for acquired spinal conditions. The distribution of vertebral shape variation within *Homo sapiens* can be conceptualized as a bell curve with an ancestral end (*left*) and a derived end (*right*). Where an individual's vertebral shape sits within this distribution has an important influence on their spinal health, according to the hypothesis. At the center of the range of variation are vertebrae that have the lineage-specific optimal shape for bipedalism and, therefore, a lower probability of developing spinal pathologies in response to the stresses of bipedal posture and gait. At the ancestral end, vertebrae differ little from those of the chimpanzees (*P. troglodytes*) and by extension from those of the common ancestor of humans and chimpanzees. People with vertebrae that fall in this part of the distribution have a heightened probability of developing intervertebral disc herniation. At the other, highly derived end of the range of variation, vertebrae exhibit exaggerated versions of our species's vertebral adaptations for bipedalism. Individuals with vertebrae that fall in this part of the distribution are more prone to develop the fatigue fractures that cause spondylolysis

fall in this "hyper-derived" part of the distribution are more prone to develop the fatigue fractures that cause spondylolysis. In other words, there is a healthy middle ground for spinal and vertebral shape, and moving away from the middle ground has consequences for spinal health—moving towards the ancestral condition for our lineage increases the probability of experiencing intervertebral disc herniation, while going beyond the middle ground increases the probability of experiencing spondylolysis. We call this the "Evolutionary Shape Hypothesis."

The Evolutionary Shape Hypothesis complements the "Neutral Zone Hypothesis" proposed by Been et al. (2019). While the lordotic angle varies considerably in modern humans, the average angle has been calculated to be 51–53° (Yang et al. 2014; Been et al. 2019). Been et al. (2019) contend that human spines with lordotic angles in the 51–53° range are in the biomechanical neutral zone, and that individuals with lordosis angles substantially lower or higher than 51–53° are at higher risk of developing spinal pathologies. The neutral zone in Been et al.'s (2019) hypothesis corresponds to the center of the range of variation in the Evolutionary Shape Hypothesis, i.e., the part of the range of variation where vertebrae that have the lineage-specific optimal shape for bipedalism are located.

A question that is obviously prompted by this attempt to place back pain in an evolutionary framework is, why have the genes underlying the shape traits that increase an individual's likelihood of developing acquired spinal conditions not been removed from our lineage through natural selection? One potential answer to this question, we think, is that not all spinal pathologies result in pain. It is not uncommon for spinal lesions to be identified in medical images of people who do not report experiencing back pain (Brinjikji et al. 2015). Thus, it is possible that the genes in question persist because in a not-insignificant percentage of individuals they are "invisible" to natural selection. Another possible answer is that even when such conditions do result in back pain, there is little impact on reproductive success. Some individuals' back pain, while persistent, is sufficiently mild that they can accomplish daily activities despite experiencing it. Others' back pain is debilitating but only happens in brief bouts and therefore does not prevent them from meeting their needs. In both situations, it is unlikely that back pain would place strong-enough selective pressures on individuals to stop them from reproducing and passing on their genes, including the genes that underlie the shape traits that increase an individual's likelihood of developing acquired spinal conditions.

Future Directions

Several next steps suggest themselves. To begin with, it would be useful to investigate the biomechanical significance of the ancestral and hyper-derived shape traits. In principle, it should be possible to accomplish this by analyzing human and great ape skeletons with a combination of dissection, 3D morphometrics, and musculoskeletal modelling. Such a study would help us understand how the shape traits increase an individual's probability of developing intervertebral disc hernias and spondylolysis. It would also provide insight into the functional anatomy of great ape vertebrae, which is something we know little about at the moment.

The Evolutionary Shape Hypothesis assumes that the shape differences between pathological and healthy human vertebrae are genetically programmed rather than the result of phenotypic plasticity responding to spinal loading regimes. There are reasons to believe this is the case. Most notably, the fact that Plomp et al. (2015, 2020) found the shape of human vertebrae with Schmorl's nodes to be similar to the shape of chimpanzee vertebrae is consistent with genetic programming but not with loading-induced phenotypic plasticity, because humans and chimpanzees share a common ancestor but have different locomotor strategies. Nevertheless, it would be helpful to establish for certain that the shape differences between Schmorl's nodes-bearing vertebrae and healthy human vertebrae are genetically programmed.

It would also be useful to identify the alleles involved in vertebral shape in humans and chimpanzees, and then investigate whether individuals with the vertebral shape associated with intervertebral disc hernias share more vertebral shape-related alleles with chimpanzees than do individuals elsewhere in the distribution of vertebral shape variation within *H. sapiens*. The same holds for the shape differences between spondylolysis-afflicted vertebrae and healthy human vertebrae. This would improve understanding of the genetic basis of specific lumbar pathologies and could open up the possibility of large-scale screening for at-risk individuals. Groundwork for this project has already been laid by research on other vertebrates (Böhmer 2017).

Another worthwhile undertaking would be to use medical imaging, geometric morphometrics, and a large sample of healthy and afflicted living humans to develop a predictive model that enables an individual's probability of developing different acquired spinal conditions to be calculated based on the shape of their vertebrae. This would allow the formulation of recommendations regarding preventative measures to reduce the likelihood of developing the relevant condition(s).

Lastly, there is reason to believe that the logic of the Evolutionary Shape Hypothesis may apply to other conditions—not only other acquired spinal conditions but also acquired conditions that affect other parts of the skeleton. The human skeleton differs in many ways from those of the great apes, and some of the differences are in regions commonly affected by acquired conditions. As such, it is possible that the link between ancestral and hyper-derived shapes and pathologies that Plomp et al. (2015, 2019, 2020) have identified in the vertebrae may hold elsewhere. The knee and hip are good candidates for such a study because they both underwent substantial changes in shape during the shift to bipedalism and are prone to acquired conditions (Watson et al. 2009). Similarly, the human shoulder differs markedly from the great ape shoulder and has a different pathology profile (Püschel and Sellers 2015).

Acknowledgments We thank Bernard Crespi and Bernard Wood for providing insightful feedback on a previous draft of this article. We also thank two anonymous reviewers and the editor for helping to improve the paper.

Funding This work was supported by the Canada Research Chairs Program (228117 and 231256), Canada Foundation for Innovation (23808 and 36801), British Columbia Knowledge Development Fund (862-804231 and 962-805808), Simon Fraser University (14518), MITACS (IT03519), the Wenner-Gren Foundation (62447), and Marie Skłodowska-Curie Actions (748200).

Declarations

Conflict of interest The authors have no competing interests to declare that are relevant to the content of this article.

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