CHAPTER 3

Acquired spinal conditions in humans: the roles of spinal curvature, the shape of the lumbar vertebrae, and evolutionary history

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3.1. Introduction

Today, back pain is both common and often serious (Hoy et al., 2014; Muthuri et al., 2018). As many as two-thirds of people in Western countries experience back pain at some point in their lives (Balague et al., 2012; Gore et al., 2012; Webb et al., 2003), and it is thought to be the single greatest contributor to disability worldwide (Buchbiner et al., 2013; Maher et al. 2017; Murray & Lopez, 2013). Because of its prevalence and the fact that it is often debilitating, back pain has substantial economic impacts (Dieleman et al., 2016; Hong et al., 2013; Webb et al., 2003). It has been estimated to cost the UK as much as £12 billion per year in direct and indirect costs (Donaldson, 2008; Maniadakis & Gray, 2000). The equivalent figure in the USA is $90 billion (Davis, 2012). Given the individual and societal impacts of back pain, improving our understanding of its causes is one of the primary foci of back pain research.

Based on the fossil and bioarchaeological records, it is clear that many of the spinal conditions that afflict people today have a long history—in some cases, a very long history. Palaeopathological studies are limited to conditions that leave traces on skeletal remains, but this still leaves a range of acquired conditions that affect the human spine, including arthritis, intervertebral disc herniation, (IDH) and spondylolysis.

Two types of arthritis that affect the spine have been identified in ancient remains. Arthritis is a general term for inflammatory and/or degenerative conditions that affect joints. Arthritis of the vertebral bodies, or spondylosis, exists in human skeletons recovered from archaeological sites dating as far back as 341,000 BP (before present) (e.g. Chapman, 1962; Bourke, 1971; Jankauskas, 1992; Jurmain, 1990; Lovell, 1994; Maat et al., 1995; Rogers et al., 1985; Strouhal & Jungwirth, 1980). It has also been diagnosed in the remains of at least two extinct hominin species, identified on lower lumbar vertebrae of a 2.14 million year old *Australopithecus africanus* specimen, Stw 431 (Odes et al., 2017; Staps, 2002; but see D'Anastasio et al., 2009), and on the cervical vertebrae of the *Homo neanderthalensis* specimen from the site of La Chapelle-aux-Saints, which dates to around 60,000 BP (Trinkaus, 1985).

The other type of spine-affecting arthritis identified in ancient remains is osteoarthritis of the zygapophyseal joints. Osteoarthritis is an arthritic condition that affects only the synovial joints and, in the spine, involves the breakdown of the synovial joints that articulate one vertebra to the next. Like spondylosis, zygapophyseal osteoarthritis is common in human skeletons recovered from archaeological sites (e.g., Bridges, 1994; Gellhorn et al., 2013; Suri et al., 2011; Waldron, 1992; Zhang et al., 2017). It is generally diagnosed through the presence of

Kimberly A. Plomp et al., *Acquired spinal conditions in humans.* In: *Palaeopathology and Evolutionary Medicine.* Edited by Kimberly A. Plomp et al., Oxford University Press. © Oxford University Press (2022). DOI: 10.1093/oso/9780198849711.003.0003

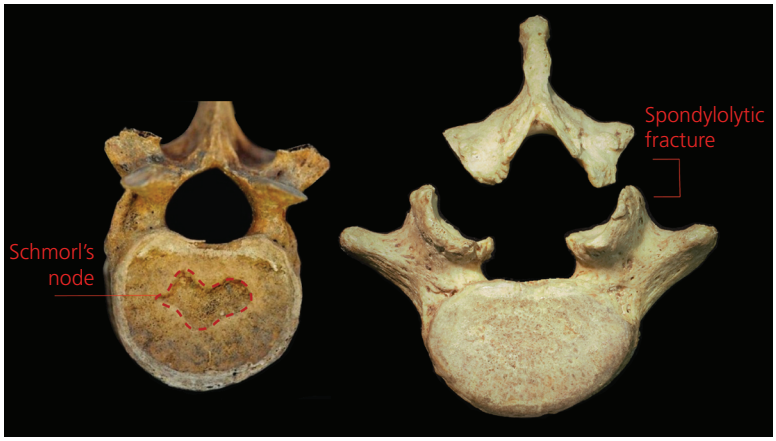

Figure 3.1 Human vertebrae exhibiting a Schmorl's node (left) and spondylolysis (right).

eburnation, or bone polishing, on the joint surface or two or more of the following joint changes: osteophytes, joint contour change and/or porosity (Rogers & Waldron, 1995). So far, the La Chapelle-aux-Saints Neanderthal specimen provides the oldest evidence of zygapophyseal osteoarthritis in the hominin fossil record (Haeusler et al., 2019).

Evidence for IDH is also found in *Homo sapiens* skeletons from archaeological sites (Mays, 2006; Plomp et al., 2012; Šlaus, 2000; Üstündağ, 2009). IDH is a condition where the gel-like substance inside the intervertebral disc, known as the nucleus pulposus, prolapses through the fibrous layers of the disc, called the annulus fibrosis. It can be identified in archaeological skeletons when the disc herniates vertically because this leaves depressions on the vertebral endplate. These depressions are called 'Schmorl's nodes' (Figure 3.1) (Schmorl & Junghans, 1971). In the hominin lineage, the oldest undisputed Schmorl's nodes have been described in the aforementioned La Chapelle-aux-Saints 1 Neanderthal specimen (Haeusler et al., 2019).

Spondylolysis also afflicted people in the past (Mays, 2007; Merbs, 1983; Waldron, 1991). It can be identified in skeletons by a uni- or bilateral separation of the neural arch from the vertebral body at the site of the pars interarticularis; in archaeological human remains the fractured ends need to show evidence of healing to be sure that the fracture is not due to post-mortem damage (Figure 3.1). The oldest evidence of spondylolysis has been identified in a late Upper Palaeolithic skeleton from Italy, Villabruna-1, which dates to 14,000 BP (Vercellotti et al., 2009, 2014).

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), some individuals suffer from back pain while others do not. Clinical studies have looked for patterns in suspected aetiological factors, including genetic predisposition, particular dietary choices, physical activities, and biochemical factors, but few patterns identified have been confirmed by subsequent studies (Hackinger et al., 2017; Nuckley et al., 2008; Nuki, 1999; Riyazi et al., 2005). 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). Unfortunately, this means that we are not much closer to understanding the causes of many spinal pathologies than we were thirty years ago.

Back pain is a complex phenomenon: it can occur in any of the five regions of the spine, i.e. the cervical, thoracic, lumbar, sacral and coccygeal regions (Figure 3.2) (Binder, 2007; Katz et al., 2003; Hoy et al., 2014; Manchikanti et al., 2002, 2004; Muthuri et al., 2018; Wild et al., 2006), and it can be chronic or acute (Patrick et al., 2014); have congenital, acquired, or idiopathic causes (Dolan et al., 2013; Giesecke et al., 2004; Modic, 1999; Taskaynatan et al., 2005); and involve soft tissue and/or bone (Dar et al., 2009; Hackinger et al., 2017; Manchikanti et al., 2002, 2004; Martin et al., 2002; Modic, 1999; Nuckley et al., 2008; Nuki, 1999; Riyazi et al.,

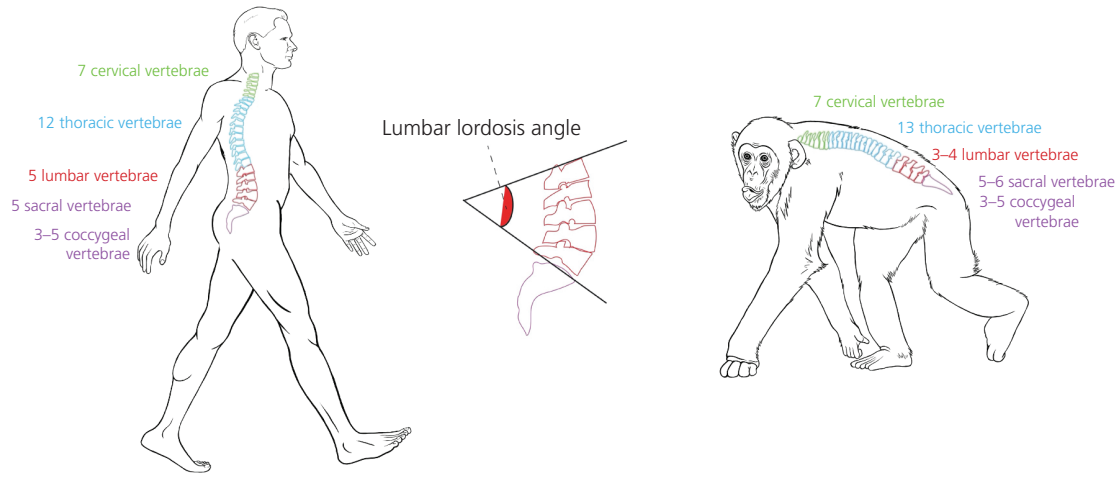

Figure 3.2 An illustration of the five regions (showing modal numbers of vertebrae in each region) and curve of a human spine (left) and chimpanzee spine (right), as well as the lumbar lordosis angle, which is calculated as the angle made between two lines, one running parallel to the superior endplate of L1 and the other running parallel to the inferior endplate of L5 (centre).

2005). This chapter focuses on acquired spinal diseases (ASDs), which are conditions of the spine that develop throughout life either through degeneration or trauma, including arthritis, IDH, and spondylolysis.

We opted to focus on ASDs, to the exclusion of lesions related to infections or development issues, because these conditions have been suggested to afflict humans due to mismatches between our spinal anatomy and our environment, and/or as trade-offs for the ability to walk on two legs (Castillo & Lieberman, 2015; Filler, 2007; Latimer, 2005; Plomp et al., 2015a). Specifically, obligate bipedalism has long been suspected to be an important aetiological factor for ASDs that afflict our species because of the types of stresses it puts on our spines (Been et al., 2019; Castillo & Lieberman, 2015; Filler, 2007; Jurmain, 2000; Keith, 1923; Latimer, 2005; Merbs, 1996; Plomp et al., 2015a). This hypothesis is based partly on the fact that humans experience ASDs far more frequently than non-human hominoids (Filler, 2007; Jurmain, 1989; Lovell, 1990; Lowenstine et al., 2016). For example, spondylosis (please note this is *not* osteoarthritis), also known as vertebral osteophytosis, is reported to affect 48–95% of humans (Cvijetić et al., 2000; Muraki et al., 2009; O'Neill et al., 1999; Prescher, 1998; Sarzi-Puttino et al., 2005). In contrast, spondylosis has been found to affect only 4% of gorillas, 5% of bonobos, and 2% of chimpanzees in non-human skeletal collections (Jurmain, 2000). Similarly, vertical IDH has been estimated to affect about 50% of modern humans with Western lifestyles but only 2% of chimpanzees and orangutans (Dar et al., 2009; Lovell, 1990). Spondylolysis is unique to humans and does not naturally occur in other animals (Ward et al., 2007).

A number of empirical studies published in the last twenty years have investigated the hypothesised relationship between bipedalism and ASDs (e.g. Been et al., 2019; Masharawi, 2012; Masharawi et al., 2007; Meakin et al., 2008, 2009; Meyer, 2016; Plomp et al., 2015a, 2019a; Scannell & McGill, 2003; Ward & Latimer, 2005; Ward et al., 2007). Collectively, these studies suggest that the relationship is mediated by the nature of the curvature of the spinal column (Been et al., 2019; Meakin et al., 2008). The various studies also suggest that the relationship is influenced by characteristics of the individual vertebrae (Masharawi, 2012; Masharawi et al., 2007; Meakin et al., 2009; Meyer, 2016; Plomp et al., 2015a, 2019a; Scannell & McGill, 2003; Ward & Latimer, 2005; Ward et al., 2007, 2010). The lumbar vertebrae are particularly important in this regard since the incidence of ASD 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, p. 4).

In this chapter, we discuss how the overall shape of the spine (as a column) and features of the lumbar vertebrae may mediate the relationship between bipedalism and some of the most common ASDs suffered by humans. We begin by explaining how spinal shape and the shape of the lumbar vertebrae relate to bipedal posture and locomotion. Next, we outline the findings of clinical studies that have found a relationship between the shapes of the spine and the lumbar vertebrae and the presence of ASDs. Subsequently, we outline palaeopathological and comparative anatomical data that also suggest that spinal curvature and the characteristics of the lumbar vertebrae impact the propensity to develop ASDs. Thereafter, we discuss recent research that suggests that the pathology-linked shapes can be understood in terms of the evolutionary history of our lineage. In the sixth section of the paper, we discuss potential biomechanical explanations for the hypothetical link between the lumbar vertebrae characteristics and IDH and spondylolysis. Finally, we close with a discussion of some potential future research directions.

3.2. Adaptations for bipedalism in the human vertebral column and lumbar vertebrae

When the human vertebral column is considered as an anatomical unit, there are two main features that are thought to be adaptations for bipedalism. One is its distinctive pattern of curvature. While great apes have a C-shaped spine, healthy adult humans have a sinuous spine (Figure 3.2; Box 3.1). The other major feature of the human vertebral column that is thought to be an adaptation for bipedalism is the number of vertebrae in the different regions of the spine (Lovejoy, 2005; Williams, 2012; Williams et al., 2013). Individuals of all the hominoid clade (and most mammals) usually have seven cervical vertebrae, but there is variation in the modal number of thoracic, lumbar, and sacral vertebrae among species (Box 3.2).

Box 3.1 The shape of the sinuous spine

The shape of the human spine is a consequence of the four pre-coccygeal regions (i.e., before the coccyx) of the spinal column having different curves (Abitbol, 1995; Been et al., 2010a, 2017; Keith, 1923; Lovejoy, 2005; Schultz, 1961; Shapiro, 1993a; Ward and Latimer, 2005; Whitcome et al., 2007). The neck or cervical region exhibits lordosis, which is a backward curvature. This results from the intervertebral discs being dorsally wedged (i.e., the discs are craniocaudally shorter at their dorsal border than at their ventral border) (Been et al., 2010a). In contrast, the upper back or thoracic region exhibits kyphosis, which is a forward curvature. This is due to ventral wedging of the vertebral bodies (i.e., the thoracic vertebrae are craniocaudally shorter at their ventral border than at their dorsal border) (Latimer and Ward, 1993). The lower back or lumbar region, like the cervical region, exhibits lordosis. Unlike in the cervical region, however, the lordosis of the lumbar region results from dorsal wedging of both the intervertebral discs and the vertebral bodies (i.e., both the discs and the vertebrae are craniocaudally shorter at their dorsal border than at their ventral border) (Been et al., 2010a). The caudal or inferior-most region of the spinal column, which is formed by the sacrum and coccyx, has a kyphotic curve. This curve results from ventral wedging of the second to fifth sacral vertebrae and all the coccygeal vertebrae and is enhanced by a ventral tilt of the cranial end of the sacrum (Antoniades et al., 2000; Cheng and Song, 2003). The four curves of the human spine are widely accepted to be functionally important (Been et al., 2010a; Latimer and Ward, 1993). They bring the centre of gravity of the body above the hips, unlike it being located ventrally in quadrupeds, and therefore allow the trunk to be balanced above the legs during bipedal walking (Latimer and Ward, 1993; Whitcome et al., 2007). The lumbar curve is particularly important in this regard (Been et al., 2010a, 2019; Latimer and Ward, 1993; Whitcome et al., 2007).

Box 3.2 Numbers of vertebrae in great apes and humans

Humans generally have twelve thoracic, five lumbar, five sacral, and three to five coccygeal vertebrae (Williams et al., 2016). The vertebral formula in great apes varies more between individuals than is typical in humans. Chimpanzees and gorillas typically have thirteen thoracic,

three to four lumbar, five to six sacral, and three to five coccygeal vertebrae, while the equivalent figures for bonobos are thirteen to fourteen, three to four, six to seven, and three to five, respectively (Williams et al., 2016). Orangutans usually have twelve thoracic vertebrae, four lumbar vertebrae, five sacral and four to six coccygeal vertebrae (Williams et al., 2016). Thus, humans tend to have a longer lumbar region than the great apes. This has been argued to result in an increased range of motion for flexion and extension (Schultz, 1953; Williams, 2012), which is especially important for maintaining the lumbar lordosis. In addition, it has been proposed that the increased space between the ribcage and the pelvis created by a longer lumbar spine, along with craniocaudally shortened iliac blades, allows for counter-rotation of the trunk relative to the hips, which helps to maintain balance during bipedal walking and running (Bramble and Lieberman, 2004; Williams et al., 2019).

Many of the traits that distinguish the lumbar vertebrae of humans from those of the great apes appear to relate to facilitating and maintaining lumbar lordosis and an upright posture. For example, the orientation of the zygapophyseal facets (Figure 3.3) is thought to be linked to vertebral slippage and rotation in the context of posture and gait (Shapiro, 1993a; Whitcome, 2012). In great apes, the zygapophyseal facets of the lumbar vertebrae are obliquely oriented, while in humans these facets are oriented more towards the sagittal plane, especially in the upper lumbar vertebrae, which has been hypothesised to resist rotation and maintain lumbar lordosis (Ahmed et al., 1990; Been et al., 2010a, Jaumard et al., 2011; Shapiro, 1993a). In addition, in humans, the distance between the zygapophyseal facets gradually increases as one moves down the lumbar spine (Latimer & Ward 1993). This has been suggested to allow for lumbar lordosis without the facets of one vertebra impinging upon the laminae or pars interarticularis of the next vertebra (Latimer & Ward, 1993; Ward & Latimer, 2005; Ward et al., 2007). Also, a larger vertebral foramen would result in the larger inter-facet distances that allow for lumbar lordosis (Latimer & Ward, 1993).

The size and orientation of the transverse processes and the attached muscles of the lumbar vertebrae also seem to play an important role in maintaining lumbar lordosis (Figure 3.3). In particular, the transverse processes of human lumbar vertebrae are shorter and more dorsally orientated than those of the great apes (Bastir et al., 2017; Jellema et al., 1993; Latimer & Ward, 1993; Plomp et al., 2019a; Ward et al., 2012; Williams & Russo, 2015). This dorsal projection results in greater invagination of the vertebral column (i.e., a ventral curve of the spinal column forward in the thorax) (Jellema et al., 1993; Latimer & Ward, 1993; Ward et al. 2012), which means that the spine is positioned forward in the thorax (Been et al. 2010a; Bogduk et al., 1992; Filler, 2007; Gómez-Olivencia et al., 2017; Sanders, 1998; Shapiro, 1993a, 2007; Whitcome et al., 2007). This increases the length of the lever arms of the erector spinae musculature and increases their ability to extend the spine, resist lateral flexion, and maintain lumbar lordosis during bipedal posture and gait (Argot, 2003; Been et al., 2010a; Benton, 1967; Gómez-Olivencia et al., 2017; Jellema et al., 1993; Latimer & Ward, 1993, 2005; Sanders, 1998; Sanders & Bodenbender, 1994; Shapiro, 1993a, 1995; Ward, 1993; Ward et al., 2012).

Several traits that distinguish the spinous processes of human lumbar vertebrae from those of great apes have likewise been argued to facilitate lumbar lordosis (Figure 3.3). In particular, the spinous processes of human lumbar vertebrae are dorsoventrally shorter (Gómez-Olivencia et al., 2013; Latimer & Ward, 1993; Meyer, 2016; Meyer et al., 2017; Plomp et al., 2019a; Schultz, 1961; Ward, 1991) and have craniocaudally short (or pinched) tips (Plomp et al., 2019a). The shortness of the spinous processes has been hypothesised to decrease the lever arms of the spinal extensor muscles and therefore limit the sagittal mobility of the spine (Argot, 2003; Gómez-Olivencia et al., 2017; Meyer, 2016; Sanders, 1998; Shapiro, 1993a, 2007; Shapiro & Kemp, 2019; Ward, 1991). 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 (Cartmill & Brown, 2017; Erikson, 1963; Gambaryan, 1974; Plomp et al., 2019a; Ritcher, 1970; Shapiro, 1993a).

There are four other traits that differentiate the human lumbar spine from the lumbar spines of the great apes. First, the bodies of the lumbar vertebrae

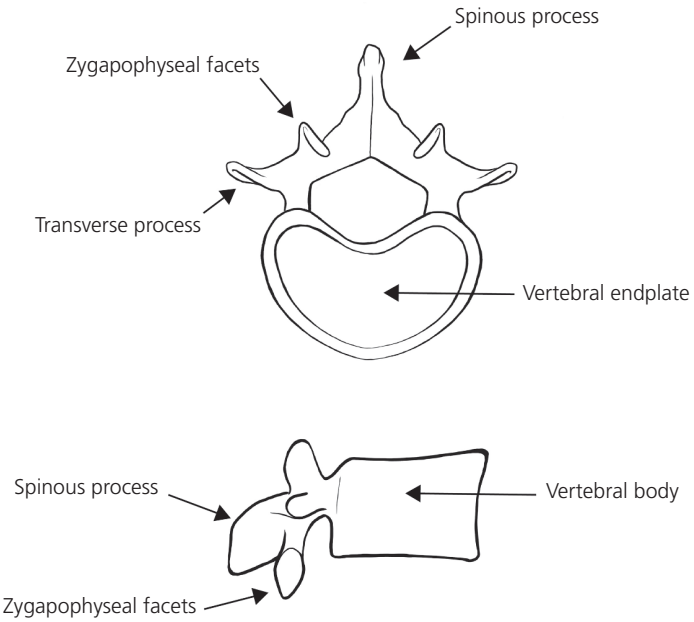

Figure 3.3 Illustration of a typical human lumbar vertebrae showing the terminology and location of vertebral elements.

of humans are dorsoventrally deeper than those of great apes (Hernandez et al., 2009; Latimer & Ward, 1993; Meyer & Williams, 2019; Plomp et al., 2015a, 2019a; Robinson, 1972). Second, the endplates of the lumbar vertebrae of humans are more heart-shaped (i.e., shorter at the midpoint of the sagittal plane compared to the coronal plane) than those of great apes, whose vertebral bodies are more circular in shape (Plomp et al. 2015a, 2019a; Robinson, 1972). Third, the vertebral bodies gradually increase in width from the first to the fifth human lumbar vertebrae (Rose, 1975; Schultz, 1953, 1961). Last, the pedicles of the last two lumbar vertebrae in the human spine are wider than those of the great apes (Figure 3.3) (Been et al., 2010b; Briggs et al., 2004; Davis, 1961; El-Khoury & Whitten, 1993; Panjabi et al., 1993; Sanders & Bodenbender, 1994; Shapiro, 1993a, 1993b; Whyne et al., 1998). All four of these traits have been hypothesised to help the vertebrae withstand the compressive load acting on the lower spine (Been et al., 2010b; Briggs et al., 2004; Davis, 1961; El-Khoury & Whitten, 1993; Hernandez et al., 2009; Latimer & Ward, 1993; Panjabi et al., 1993; Plomp et al., 2012, 2015a,b, 2019b; Rose, 1975; Shapiro, 1991, 1993a; Sanders & Bodenbender, 1994; Whyne et al., 1998).

3.3. Clinical evidence for an impact of spinal and vertebral shape on spinal health

Many of the clinical studies that have investigated the relationship between vertebral shape and spinal health have focused on lumbar lordosis (e.g., Been & Kalichman, 2014; Been et al., 2019; Keller et al., 2005; Scannell & McGill, 2003; 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 vertebra and a line running parallel to the first sacral endplate (Figure 3.2), 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 humans (Been & Kalichman, 2014; Zlolniski et al., 2019) and this variation is associated with the propensity to develop ASDs (Been et al., 2019; Keller et al., 2005; Scannell & McGill, 2003).

One ASD that has been linked with the lordotic angle is osteoarthritis of the zygapophyseal joints. Osteoarthritis is a breakdown of synovial joints,

which in the spine are the zygapophyseal and costovertebral joints. Clinically, osteoarthritis preferentially affects individuals with pronounced lumbar lordosis (Roussouly & 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), which may be related to the increased lordosis. 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 & Pinheiro-Franco, 2011; Weinberg et al., 2017).

Clinical studies have also suggested that a large lordotic angle may contribute to spondylolysis, which is a cleft in the neural arch caused by a fatigue fracture at the site of the pars interarticularis (Hu et al., 2008). It is particularly common in athletes (Iwamoto et al., 2004), with one study of 100 American adolescent athletes with low back pain finding that 47% had spondylolysis (Micheli & Wood, 1995). Using clinical radiographs, Roussouly and colleagues (2006) found that unusually pronounced lordosis was associated with spondylolysis. In a similar vein to the aforementioned explanations for spinal osteoarthritis, they proposed that a large lordotic angle increases the direct contact between the neural arches of the lumbar vertebrae and ultimately causes the fractures that lead to spondylolysis (Roussouly et al., 2006).

Spondylolysis also has been linked with the shape of the zygapophyseal facets. Specifically, it has been 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 mentioned, the size, shape and orientation of the vertebral facets are associated with the curvature of the spine (Shapiro, 1993a; Whitcome, 2012). In the lumbar spine, the zygapophyseal facets are oriented towards the sagittal plane and become increasingly coronally oriented moving down the lumbar spine, which likely helps to maintain lumbar lordosis (Ahmed et al., 1990; Been et al., 2010a, Jaumard et al., 2011; Latimer & Ward, 1993; Shapiro, 1993a). On this basis, it is thought that the flatness and exaggerated coronal orientation of the facets identified in L4 and L5 vertebrae with spondylolysis may not provide adequate facilitation for, and may instead restrict, the large lordotic angle that is also associated with the condition (Roussouly et al., 2006).

While a number of clinical studies suggest that having an unusually pronounced lumbar lordosis may increase the likelihood of developing zygapophyseal osteoarthritis and spondylolysis, there is also clinical 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 IDH have significantly smaller lordotic angles than those with healthy spines (Barrey et al., 2007; Ergun et al., 2010; Yang et al., 2014). Specifically, these studies have found that individuals with degenerative changes to their discs had an average lordotic angle of 40° while those with disc herniations had 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. Analyses by Been and colleagues (2010a) and Yang et al. (2014) indicate that the average lordotic angle in healthy humans is 51–53°.

Clinical studies have identified two other traits that appear to be correlated in humans with IDH, a condition where the gel-like substance inside the intervertebral disc prolapses through the fibrous layers of the disc. One of the traits was identified by Harrington and colleagues (2001), who used computed tomography (CT) scans of 97 patients to measure vertebral endplate dimensions and found that individuals with IDH tended to have endplates that are more circular in shape, compared to the more heart-shaped endplate in healthy vertebrae. The other trait was recognised by Pfirrmann and Resnick (2001), who performed an analysis of thoracic and lumbar vertebrae and intervertebral discs from 128 cadavers and discovered that intervertebral disc hernias affected vertebrae with

flatter endplates significantly more frequently than vertebrae with more concave endplates.

Based on these clinical studies, it appears that lumbar lordosis plays an important role in spinal health, with a more-pronounced-than-normal lordotic angle possibly leading to spondylolysis and a less-pronounced-than-normal angle potentially increasing the likelihood of IDH. Been and colleagues (2019) proposed what they called the 'Neutral Zone Hypothesis' to explain this pattern. They argued that there is a 'neutral zone' for the lordotic angle in the human spine and that deviations from this zone increase the chances of developing spinal pathologies. They based the neutral zone on a previous study in which members of the same team had calculated the average angle of healthy human spines to be about 51° (Been et al., 2010a). They argued that an individual with a lordosis angle that is at least 10° lower or higher than the average of 51° is at risk of developing spinal pathologies (Been et al., 2019).

3.4. Palaeopathological and comparative anatomical evidence for an impact of spinal and vertebral shape on spinal health

In a similar vein to the clinical studies outlined in Section 3.3, palaeopathological data has been used to investigate whether vertebral shape variation is an aetiological factor in the development of Schmorl's nodes. Plomp and colleagues (2012, 2015b) compared the 2D shape of the superior planar surface of thoracic and lumbar vertebrae with and without Schmorl's nodes from medieval and post-medieval English populations. They found that human vertebrae with Schmorl's nodes differed in shape from those without the lesions. Their analyses indicated that, based on shape traits, vertebrae with Schmorl's nodes could be identified with an accuracy rate of 69–81%. Given these findings, the authors proposed that vertebral shape may be an important factor in the aetiology of IDH and therefore in the development of Schmorl's nodes. They were able to confirm that the superior endplates of vertebrae with Schmorl's nodes tended to be more circular than those of healthy vertebrae. This aligns with the clinical study performed by Harrington and colleagues (2001). In addition, Plomp and colleagues (2012, 2015b) found that vertebrae with Schmorl's nodes have shorter pedicles and laminae and smaller vertebral foramina than human vertebrae without Schmorl's nodes.

Several palaeopathological and comparative studies investigating the relationship between the condition and vertebral shape have focused on spondylolysis. Similar to the clinical evidence, one study concluded that an unusually high lordotic angle makes individuals more susceptible to developing spondylolysis (Roussouly et al., 2006). Masharawi and colleagues (2007) compared the dorsal and ventral heights of L5 and found that spondylolytic vertebrae tend to have bodies that are more dorsally wedged (i.e., the ventral border of the vertebral body is craniocaudally taller than the dorsal border) than healthy vertebrae. More pronounced wedging should result in a larger lordotic angle, so this result aligns with the findings of Roussouly and colleagues' (2006) clinical study. In addition, Ward and colleagues (2005, 2007, 2010) found a correlation between spondylolysis and a trait that is thought to be related to lumbar lordosis—specifically, that individuals with spondylolysis tend to have reduced transverse spacing between the zygapophyseal facets of adjoining vertebrae compared to those without spondylolysis. Transverse spacing between the facets increases as one moves down the human lumbar spine and this is thought to allow for lumbar lordosis. Given this, Ward and colleagues (2007, 2010) hypothesised that reduced mediolateral spacing leads to the articular processes of one vertebra directly contacting the pars interarticularis of the subjacent one, leading to spondylolysis (Ward & Latimer, 2005; Ward et al., 2007, 2010).

3.5. Evolutionary origins of vertical intervertebral disc herniation and spondylolysis

Based on the results of the clinical, comparative, and palaeopathological studies that have been carried out to date, it appears that variation in vertebral shape influences an individual's propensity to

develop a number of ASDs. So, why do some people have vertebral shapes that predispose them to such conditions? Three recent studies have attempted to answer this question by investigating the evolutionary origins of vertebral shape variation in relation to two lesions that have been linked with vertebral shape in clinical, comparative, and palaeopathological studies: Schmorl's nodes and spondylolysis (Plomp et al., 2015b, 2019b, 2020).

Plomp and colleagues (2015a) compared the 2D shape of human final thoracic and first lumbar vertebrae from archaeological populations with those of chimpanzees and orangutans. They used chimpanzee and orangutan vertebrae as comparators to identify evolutionary traits because although it is accepted that humans and chimpanzees shared a common ancestor to the exclusion of other apes, the locomotor behaviour of the common ancestor is debated. The most popular hypotheses are that the ancestor used either knuckle-walking similar to modern chimpanzees, bonobos, and gorillas, or quadrumanous climbing (i.e., using all four feet to grasp branches), such as performed by modern orangutans (Richmond et al., 2001; Thorpe et al., 2007). Plomp et al. (2015a) divided the humans into two groups: one comprising individuals who had Schmorl's nodes, and the other consisting of individuals with no visible spinal pathologies. They found that the vertebrae of humans with Schmorl's nodes were closer in shape to those of chimpanzees than were the healthy human vertebrae. Human vertebrae with Schmorl's nodes and chimpanzee vertebrae were found to have more circular vertebral bodies; shorter, narrower pedicles; and relatively smaller vertebral foramina. The authors argued that because chimpanzees, bonobos and modern humans share an ancestor to the exclusion of all other living species, any vertebral traits that chimpanzees and humans have in common are most likely inherited from their common ancestor. Given this, they asserted, it is reasonable to suppose that humans with Schmorl's nodes experience IDH because their vertebrae were closer to the ancestral shape for the hominin lineage. This ancestral shape, they continued, is not as well adapted to withstand the stresses placed on the spine during bipedalism and thus, increases the likelihood of disc herniations. Plomp and colleagues (2015a) called this the 'Ancestral Shape Hypothesis'.

Subsequently, Plomp and colleagues (2019b) tested the Ancestral Shape Hypothesis with 3D shape data from the last two thoracic and first lumbar vertebrae of modern humans with and without Schmorl's nodes, chimpanzees and several extinct hominins. As before, they found that modern human vertebrae with Schmorl's nodes shared more similarities in shape with chimpanzee vertebrae than did healthy modern human vertebrae. They also found that the human vertebrae with Schmorl's nodes were closer in shape to the vertebrae of a number of extinct hominins, including Sts 14 (*Australopithecus africanus*), MH1 (*Australopithecus sediba*), SK 853 (*Paranthropus robustus*), UW 101-1733 (*Homo naledi*), Kebara 1 (*H. neanderthalensis*), and Kebara 1 (*H. neanderthalensis*), than were the healthy human vertebrae. They argued that these results provide further support for the Ancestral Shape Hypothesis because they demonstrate that the human vertebrae with Schmorl's nodes do indeed lie towards the ancestral end of the range of shape variation in *H. sapiens*.

The following year, Plomp and colleagues (2020) investigated whether the propensity to develop another spinal condition is affected by individuals' location in the ancestral-to-highly derived spectrum of vertebral shape variation. Building on the putative link between spondylolysis and large lumbar lordosis (Masharawi et al., 2007; Roussouly et al., 2005; Ward et al., 2005, 2007), they hypothesised that spondylolytic vertebrae may have the opposite shape problem to those with Schmorl's nodes. Such vertebrae may, they suggested, exhibit shape traits that are exaggerated adaptations for bipedalism. They called this the 'Overshoot Hypothesis' (Plomp et al., 2020).

To test this, they compared the 3D shape of final lumbar vertebrae of humans, chimpanzees, gorillas, and orangutans. The humans were divided according to whether they had bilateral spondylolysis, Schmorl's nodes on any vertebrae in the spine, or no vertebral lesions. The authors found that, as predicted, the spondylolytic human vertebrae shared fewer similarities in shape with those of great apes than did the healthy human vertebrae. Again, as predicted, they found that vertebrae of humans

with Schmorl's nodes sat on the opposite end of the range of variation from the spondylolytic human vertebrae, and that healthy human vertebrae fell between the two groups of pathological vertebrae. Plomp and colleagues (2020) argued that this means that spondylolytic vertebrae show fewer similarities in shape with the vertebrae of great apes than do either healthy human vertebrae or those with Schmorl's nodes. Furthermore, the main ways that spondylolytic vertebrae differ from the other three groups of vertebrae is that they tend to have vertebral bodies that are more dorsally wedged, narrower inter-pedicle distances, more dorsally projecting pedicles, and narrower inter-facet distances. Plomp and colleagues (2020) concluded that their results support the Overshoot Hypothesis.

The findings of these comparative studies suggest that the prevalence of some important ASDs in modern humans is partially explained by the evolution of vertebral shape variation and how well different vertebral shapes withstand the stresses placed upon the bipedal spine. They imply that we can visualise human vertebral shape variation as a bell-shaped distribution (Figure 3.4). Vertebrae at one end of the distribution display traits that are similar to those of chimpanzee vertebrae and, by extension, the vertebrae of the common ancestor of the hominins. These vertebrae are prone to one type of ASD: IDH. Vertebrae at the other end of the bell-shaped distribution are characterised by traits that are basically exaggerated versions of some of the key vertebral adaptations for bipedalism in humans. These vertebrae are prone to a different type of ASD: spondylolysis. Between the two extremes are vertebrae that are at, or close to, 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. Importantly, this hypothesis implies that where an individual's vertebral shape sits on this evolutionary spectrum likely influences their spinal health. This hypothesis, which we will refer to hereafter as the 'Evolutionary Shape Hypothesis', clearly overlaps with

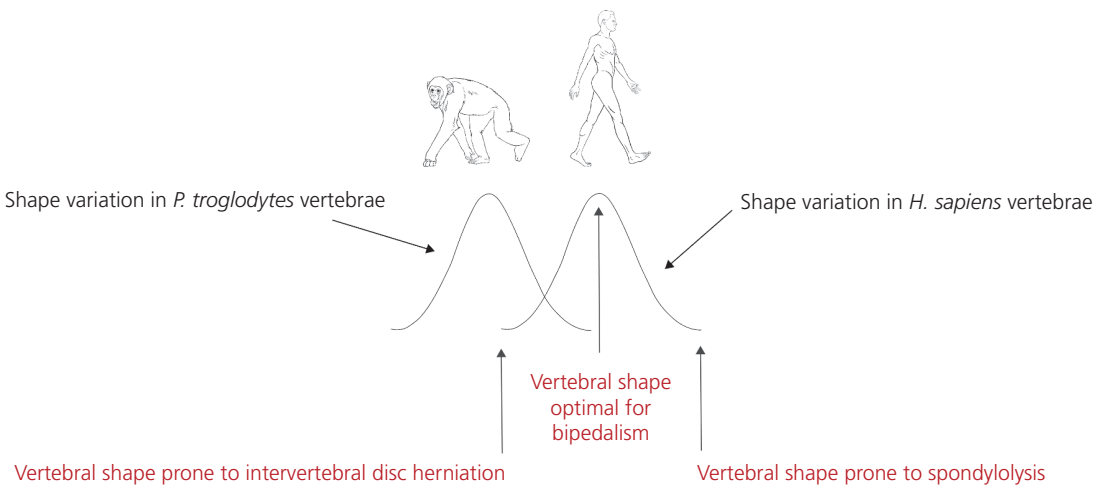

Figure 3.4 The logic of the Evolutionary Shape Hypothesis for back pain. The distribution of vertebral shape variation within humans can be conceptualised as a bell-curve with an ancestral end (left) and a derived end (right). According to the hypothesis, where an individual's vertebral shape sits within this distribution has an important influence on their spinal health. At the centre of the range of variation are vertebrae that have the lineage-specific optimal shape for bipedalism and, therefore, a lower probability of developing spinal disease in response to the stresses of bipedal posture and gait. At the ancestral end, vertebrae differ little from those of 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 vertical intervertebral disc herniation that can lead to Schmorl's nodes. At the other, highly derived end of the range of variation, vertebrae exhibit exaggerated versions of our species' 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.

Been and colleagues (2019) Neutral Zone Hypothesis (Section 3.3). Been and colleagues (2019) neutral zone of spinal curvature corresponds to the area under the middle of the bell-curve in Figure 3.4—the area where individuals have vertebrae that are at, or close to, the lineage-specific optimum shape for facilitating bipedal posture and gait.

3.6. Potential biomechanical explanations for links between vertebral traits and acquired spinal diseases

Table 3.1 summarises the vertebral traits found to correlate with the presence of IDH as indicated by Schmorl's nodes, and the vertebral traits that have been found to correlate with the occurrence of spondylolysis. This section outlines some biomechanical hypotheses that attempt to explain how the two sets of traits give rise to the pathologies.

To begin with, vertebrae with Schmorl's nodes tend to have bodies that are less dorsally wedged than the bodies of healthy vertebrae (Plomp et al., 2019a). In principle, this should result in a smaller lordotic angle and therefore a straighter spine (Been et al., 2019). Having a less-pronounced lordotic angle can be expected to influence how the spine absorbs compressive loads during bipedalism (Farfan, 1995; Gracovetsky & Iacono, 1987; Whitecome et al., 2007), as well as increase the load acting on the intervertebral discs (Wei et al., 2013). A straighter lumbar spine should result in a more ventral placement of compressive loads, meaning that most of the loading would occur on the vertebral bodies and intervertebral discs, rather on the bodies, discs, and neural arches (Adams et al., 1994). This may result in an increased propensity to develop disc herniations, because biomechanical tests showed that herniations often occur when the discs are subjected to both compressive and shearing forces (Cholewicki & McGill, 1996). In other words, lumbar vertebrae with bodies that are less dorsally wedged would result in a smaller lordotic angle, which may not be biomechanically well suited to withstand the compressive loading placed on the lower spine during bipedalism (Plomp et al., 2019a).

Another Schmorl's nodes-associated trait of the vertebral body may influence how the vertebrae withstand compressive loads. Lumbar vertebrae with Schmorl's nodes have been found to have more circular vertebral bodies compared to healthy vertebrae, which are more heart-shaped (Plomp et al., 2015a, 2019b). This is significant because more circular endplates can be expected to have a larger diameter compared to more heart-shaped endplates, and increased disc diameter has been argued to foster disc herniation. The explanation for this is LaPlace's Law (Letić, 2012), which states that the ability of a fluid-filled tube to withstand tension decreases with increasing radius. This means that an intervertebral disc/vertebral body that is more circular may be less able to withstand the compressive loads acting

Table 3.1 Summary of the vertebral traits associated with Schmorl's nodes and spondylolysis

	Compared to healthy human vertebrae
Vertebrae with Schmorl's nodes have:	Vertebral bodies that are less dorsally wedged Vertebral bodies that are more circular compared to healthy vertebrae, which are more the heart-shaped Pedicles and laminae tend to be short Transverse processes that are longer and project more laterally Spinous processes that are longer and more cranially oriented Vertebral bodies that are more dorsally wedged resulting in a more pronounced lordotic angle
Vertebrae with spondylolysis have:	Pedicles that project more dorsally Narrower inter-pedicle distances Zygapophyseal facets that are more caudally located Narrower inter-facet distances Inferior endplates with deeper concavities

on the bipedal spine than a heart-shaped vertebral body (Harrington et al., 2001; Plomp et al., 2012, 2015a).

The pedicles and laminae also play an important role in withstanding compressive loads by acting as structural buttresses for the vertebral body (Adams et al., 1994; El-Khoury & Whitten, 1993; Whyne et al., 1998). Plomp and colleagues (2012, 2015a, 2019b) found that the pedicles and laminae of lumbar vertebrae with Schmorl's nodes tended to be relatively short, and suggested that this may make them less able to buttress against loads than the pedicles and laminae of healthy human vertebrae.

The transverse process traits found to be associated with Schmorl's nodes probably relate to the stability of the lumbar spine rather than withstanding loads. For example, comparative analyses have found that mediolaterally longer transverse processes allow for lateral flexion in the lower spine (Argot, 2003; Sanders, 1998; Shapiro, 1993a). Thus, the longer, laterally projecting transverse processes identified in vertebrae with Schmorl's nodes may not provide adequate stability during bipedalism. In addition, transverse processes that project more laterally may be less able to maintain lumbar lordosis than those that project dorsally (Been et al., 2017; Bogduk et al., 1992; Filler, 2007; Sanders, 1998; Sanders & Bodenbender, 1994; Whitcome et al., 2007). Given this, the longer, laterally projecting transverse processes of vertebrae with Schmorl's nodes may increase dorsomobility and result in a lumbar spine that is less stable during bipedalism. The longer, cranially oriented spinous processes of humans with Schmorl's nodes may also cause spinal instability. Specifically, it has been argued that long cranially oriented spinous processes may allow for a greater amount of dorsal mobility in the spine, while short, caudally oriented spinous processes are associated with a less mobile and more stable spine (Argot, 2003; Gómez-Olivencia et al., 2017; Meyer, 2016; Sanders, 1998; Sanders & Bodenbender, 1994; Shapiro, 1993a, 1995, 2007; Shapiro et al., 2005; Ward, 1991). Considering this, Plomp and colleagues (2019b) suggested that the longer, cranially oriented spinous processes of people with Schmorl's nodes may predispose individuals to IDH.

Regarding the traits associated with spondylolysis, Plomp and colleagues (2020) found that L5 vertebrae with spondylolysis have more pronounced dorsal wedging, which can be expected to result in a hyper-lordotic lumbar spine (Masharawi et al., 2007; Plomp et al., 2020). Exaggerated lordosis has been suggested to increase direct contact between the neural arches of the lumbar vertebrae and this has been posited to result in the fatigue fractures that cause spondylolysis (Masharawi et al., 2007). The increased contact between the neural arches is exacerbated by four other traits associated with spondylolysis: narrower inter-pedicle distances, dorsal projection of the pedicles, narrower inter-facet distance, and caudally located zygapophyseal facets (Plomp et al., 2020; Ward et al., 2005, 2007, 2010). The first three of these traits can be expected to result in a mediolaterally narrower neural arch width and therefore, in smaller inter-facet distances (Plomp et al., 2020; Ward et al., 2005, 2007, 2010). Unlike quadrupedal apes, humans have a consecutive increase in inter-facet distances from the top to the bottom of the lumbar spine, which is thought to allow for adequate spacing between the facets in the presence of lumbar lordosis (Latimer & Ward, 1993). Thus, it has been hypothesised that the reduced inter-facet spacing caused by the mediolaterally narrower neural arch width may lead to the articular processes of one vertebra directly contacting the pars interarticularis of the subjacent vertebra, causing the fatigue fracture and, ultimately, spondylolysis (Plomp et al., 2020; Ward et al., 2005, 2007, 2010). The fourth trait—caudally located zygapophyseal facets—can be expected to result in crowding of those joints (Plomp et al., 2020). This has been suggested to increase the contact between the inferior facets of L4 and the pars interarticularis of L5, causing fatigue fractures that can eventually lead to spondylolysis (Plomp et al., 2020).

The last trait associated with spondylolytic vertebrae is a deeper concavity of the inferior endplate (Plomp et al., 2020). Based on clinical studies, this trait may influence the ability of the vertebrae to disperse compressive stress. For example, Liu and colleagues (2007) found that vertebrae with endplates with shallower concavities were better suited to withstand compressive strains and this led to

a decrease in the amount of stress placed on the zygapophyseal facets and neural arch. With this in mind, Plomp and colleagues (2020) posited that the increased concavity of vertebral bodies may result in increased loading placed on the posterior vertebral elements, including the pars interarticularis, and ultimately increase the risk of developing spondylolysis.

3.7. Future directions

There are a number of obvious potential avenues for research in the future. One is to evaluate the biomechanical hypotheses outlined in Section 3.6 by analysing human and great ape vertebrae with a combination of dissection, 3D morphometrics, and musculoskeletal modelling. Such a study would help us understand how the 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, about which we currently know very little.

A second possible project 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 spine 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 condition(s).

A third worthwhile endeavour would be to identify the alleles involved in vertebral shape in humans and chimpanzees, and then investigate whether individuals with the vertebral shape associated with IDH share more vertebral shape-related alleles with chimpanzees than do individuals elsewhere in the distribution of vertebral shape variation within humans. This would improve understanding of the genetic basis of specific lumbar diseases and could open up the possibility of large-scale screening for at-risk individuals. The foundations for this project have already been laid by work on other vertebrates (Böhmer, 2017).

Finally, the logic of the Evolutionary Shape Hypothesis may also apply to other acquired diseases affecting the human skeleton—not only in the spine, but also those 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 afflicted by acquired conditions. As such, it is possible that the link between ancestral and hyper-derived shapes and diseases identified by Plomp and colleagues (2015a, 2019b, 2020) 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 also prone to acquired diseases (Watson et al., 2009). Similarly, the human shoulder differs markedly from that of the great apes and has a different pathology profile (Püschel and Sellers, 2015).

3.8. Conclusions

This chapter has revisited Keith's (1923) classic hypothesis that the stress placed on our spines by our unique mode of locomotion explains why we are so commonly afflicted with back problems. Specifically, we reviewed and synthesised evidence indicating that the relationship between bipedalism and some important ASDs is mediated by the degree of curvature of the lumbar spine and certain shape traits of the lumbar vertebrae. Subsequently, we outlined a revised version of Keith's (1923) Evolutionary Shape Hypothesis, which states that human vertebral shape variation should be viewed as a spectrum, where vertebrae at one end are similar to great ape and fossil hominin vertebrae and vertebrae at the other end exhibit exaggerated adaptations for bipedalism. According to the Evolutionary Shape Hypothesis, where an individual's vertebrae sit on this evolutionary shape spectrum has an important influence on their spinal health. If a person has vertebrae that lie at the ancestral end, they have a higher probability of developing intervertebral disc hernias. Conversely, if their vertebrae lie at the other, highly derived end of the spectrum, they have a greater likelihood of developing spondylolysis.

The Evolutionary Shape Hypothesis not only provides novel insights into what causes back problems, but also has the potential to inform how clinicians manage people with common spinal diseases. As discussed in Section 3.7, the Evolutionary Shape Hypothesis could form the basis of a predictive

model for identifying individuals who are at risk of developing different ASDs. In principle, it should be possible to use medical imaging technology, for example, magnetic resonance imaging (MRI) and computerised tomography (CT), alongside geometric morphometrics to analyse a living individual's lumbar vertebrae and assign them to risk categories for IDH and spondylolysis. It should then be possible to devise behavioural strategies that would reduce chances of developing the ASD for which they are at risk, for example, avoiding certain sports. This would represent a substantial step forward in the management of spinal health and therefore of back pain.

More generally, this chapter demonstrated the benefits and applications of palaeopathological and palaeoanthropological data for evolutionary medicine (EM), which is a fast-expanding field that involves the application of Darwinian thinking to medical problems (Nesse & Williams, 1994). Since EM solidified as a field in the early 1990s, a considerable amount of research has illustrated the benefits of looking at health issues with an evolutionary lens. However, so far there have been few attempts to analyse musculoskeletal problems within the framework of EM and consequently very little use of palaeopathological or palaeoanthropological data in EM. This chapter illustrates not only that it is possible to shed new light on musculoskeletal problems with an EM approach, but also that palaeopathological and palaeoanthropological data can be extremely useful for this endeavour.

Furthermore, this chapter demonstrated that palaeopathology can benefit from drawing on evolutionary theory. Despite palaeopathology's ability to provide insight into the health of past peoples, there are a few issues that, historically, have decreased its relevancy to medical professionals. One major criticism levelled at palaeopathology is that it is too focused on individual and unique case studies, rather on hypothesis-driven, population-based research (Rühli et al., 2016). EM offers a robust theoretical framework that can address this criticism. If the palaeopathological and comparative data outlined in this chapter had not been evaluated with an evolutionary perspective, they would have only provided information on how vertebral shape correlates with the presence of ASDs. Instead, interpreting the data within an evolutionary framework allowed for the development of a novel, productive causal hypothesis for ASDs. In our view, this represents a major advance, and one that can probably be replicated with many of the other skeletal diseases on which palaeopathologists work.

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